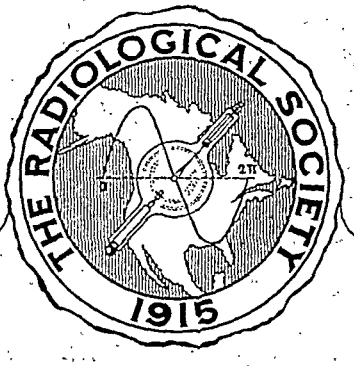
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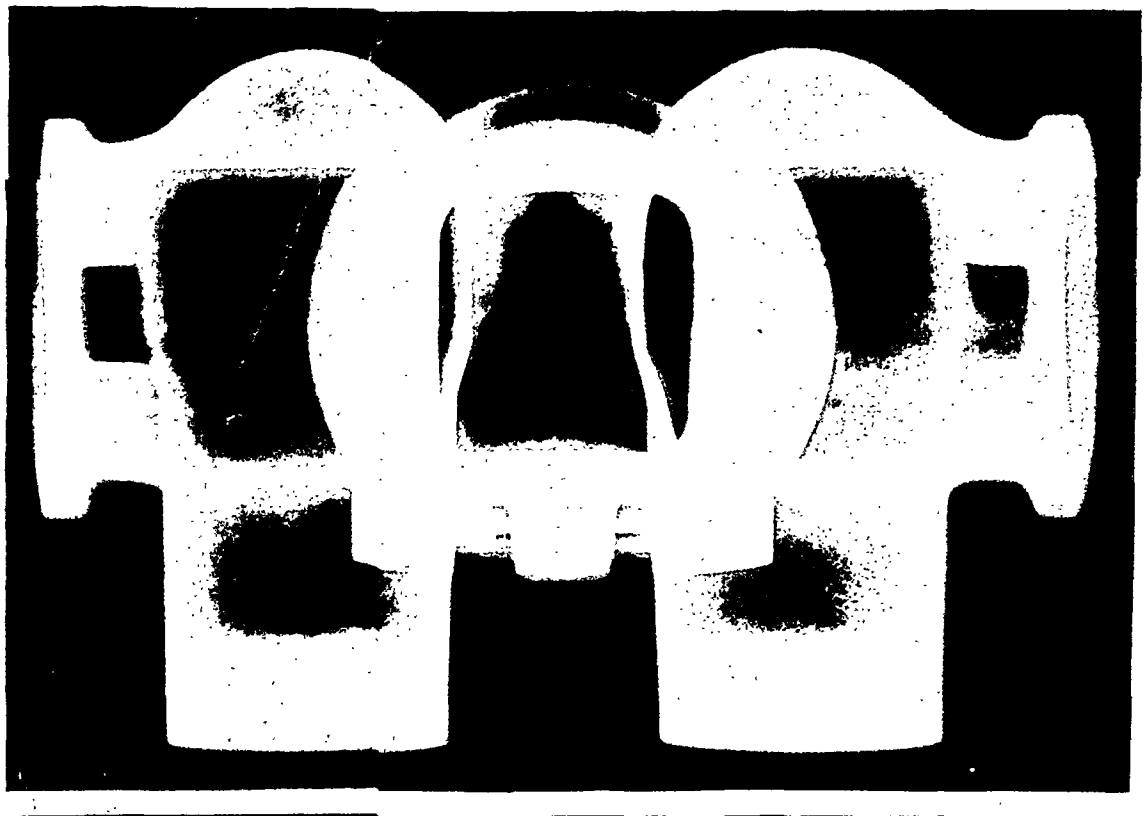


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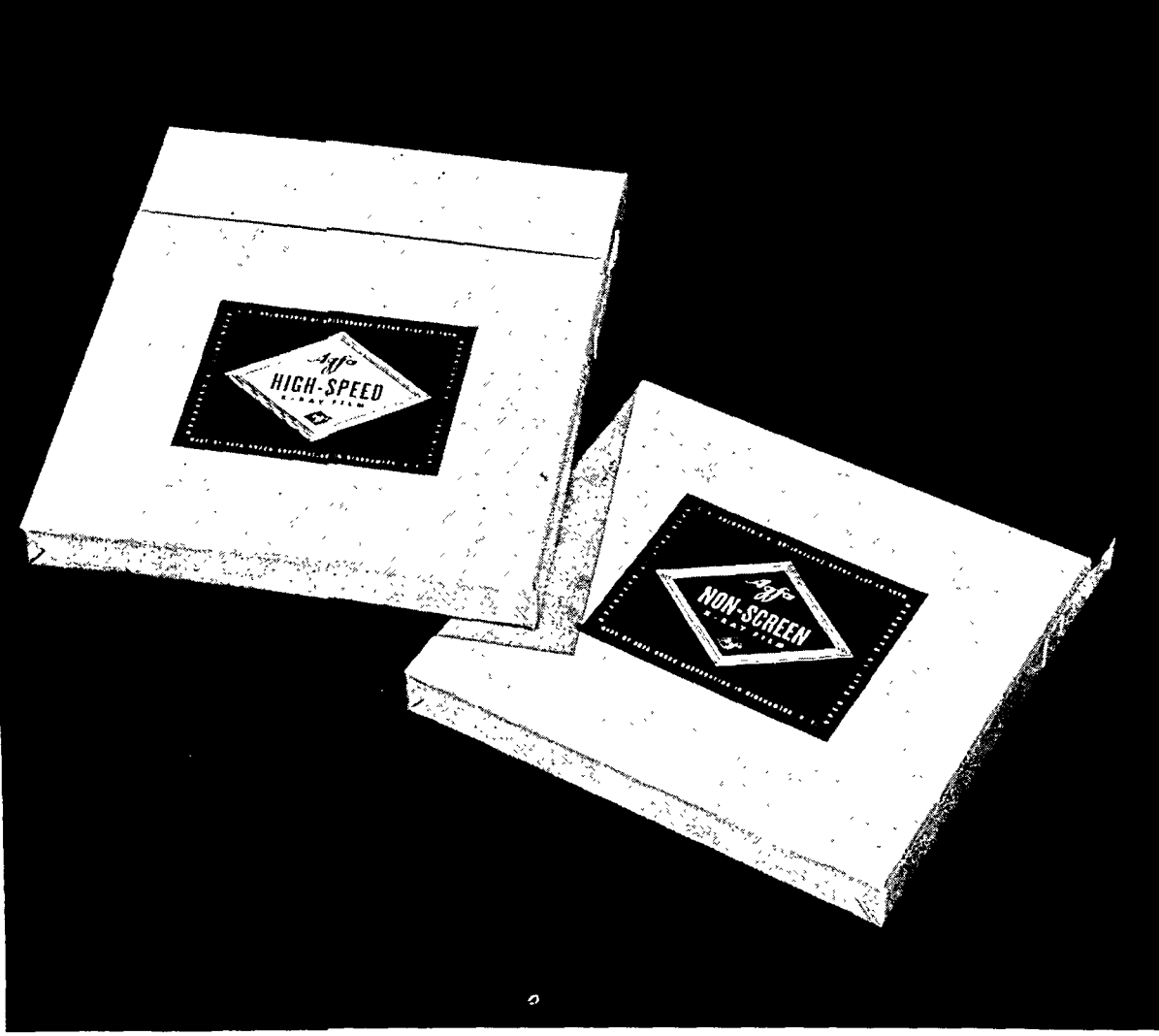
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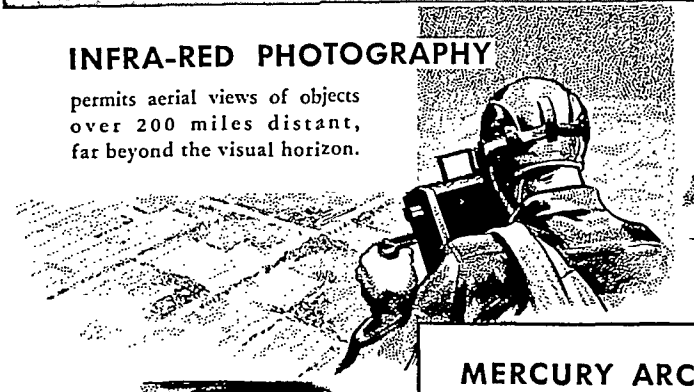
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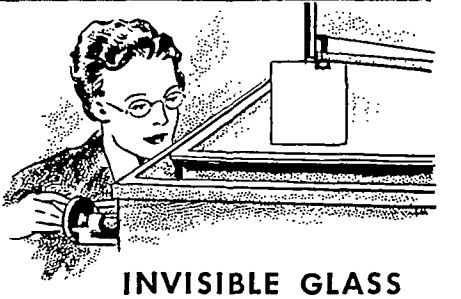
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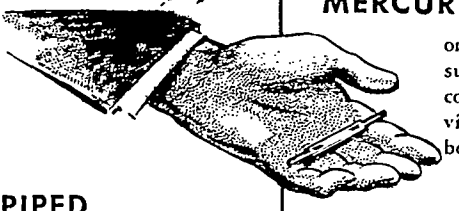


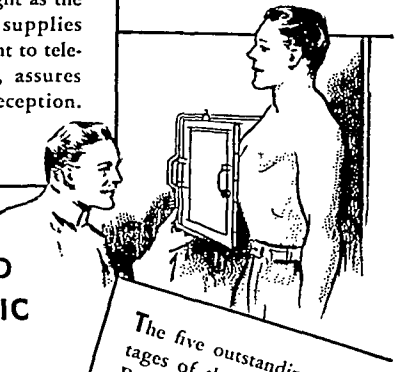
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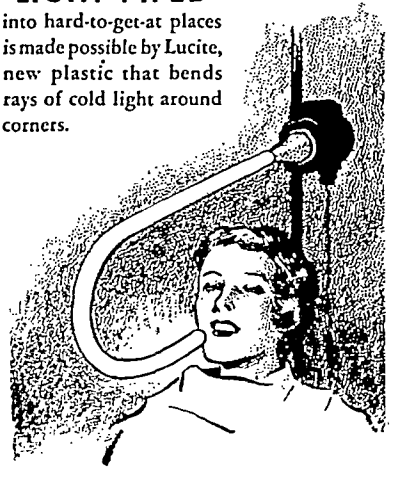
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ANTEPARTUM ROENTGENOGRAPHIC DIAGNOSIS OF FETUS PAPYRACEOUS[1]

By ALBERT C. JOHNSTON, M.D , *Berlin, N. H., and* MERRILL C. SOSMAN, M.D., *Boston, Mass.*

From the Department of Roentgenology, Peter Bent Brigham Hospital

FETUS papyraceous is easily diagnosable by antepartum roentgenograms at any time between the death of the twin fetus and delivery. The death of one fetus usually occurs between the third and fifth month of pregnancy. A careful search of the literature, however, reveals but one case correctly diagnosed roentgenographically before delivery (19). Kerr and Rypins (10) demonstrated the fetus papyraceous roentgenographically antepartum, but it was incorrectly interpreted until after delivery. We are reporting herewith a second case so diagnosed, and four other cases of fetus papyraceous in which the opportunity to make the diagnosis roentgenographically was not offered.

Fetus papyraceous (or compressus) is the dead, mummified, and flattened fetus associated with a living twin. Mosher (13) made a complete survey of the literature in 1917 and found only 88 cases reported up to that time. Twenty-six of the reported cases were from America.

Since 1917, we have found 16 additional cases in the literature which, with the five herewith reported, brings the total to 109. Four of the recent reports concern cases of triplets with fetus papyracii (3, 5, 14, 21), and one of them (Beierlein, 3) collected 20 cases of triplet pregnancy with twin fetus papyracii and added one of his own. Beierlein stated that the diagnosis could but rarely be made antepartum and expressed a doubt that the fetus papyraceous would be demonstrable roentgenographically. The incidence of fetus papyraceous, according to Petzold (16), from the Frauenklinic in Vienna, is six times as common in single ovum twins, with double amnii and a single chorion as in double ovum twins. It more frequently occurs in primipara.

The history may give a clinical suggestion of what has happened. In many of the reported cases, there is a history of bloody discharge or actual bleeding at the time of fetal death, which usually occurs between the fourteenth and twentieth week of pregnancy. A bloody discharge is common to so many conditions during pregnancy that one would not suspect the death of a twin fetus unless the second sign were also present. This is the presence of an enlarged uterus, larger by palpation and measurements than one would expect from the duration of normal single pregnancy. The third clinical hint as to what is going on is the regression of the larger-

[1] Presented in part before the New England Roentgen Ray Society, Feb. 18, 1938.

than-normal uterus to normal size after the bleeding occurs, with subsequent normal development, as would be expected in the usual single pregnancy. The diagnosis of fetus papyraceous has been reported to have been made several times before delivery by vaginal palpation of the small fetal structures protruding ahead of the presenting part of the living fetus. The most accurate method, however, is the demonstration of the small but well ossified skeleton of the fetus papyraceous in the roentgenogram (Fig. 1).

The cause of death of one of the twins may not be evident, but it is assumed that the fetal circulation is shut off. This is based on the finding of an infarcted atrophic or fibrosed placenta belonging to the dead fetus. When the blood supply is shut off and the fetus dies, the liquor amnii is resorbed and the dead fetus becomes compressed between the amniotic sac of the living twin and the uterine wall. The removal of the liquor amnii with its enzymes, which play an important rôle in the production of maceration, allows the fetus in its dry aseptic state to mummify. Complete dissolution of the dead fetus is possible only in the early weeks of pregnancy and cannot occur after it has reached any considerable proportions (Williams, 23). The usual sequence of events in single pregnancy with death of the fetus is maceration and abortion. Only in multiple pregnancy does the mummification occur which results in fetus papyraceous. Occasionally an extra-uterine fetus may die and become calcified—fetus, placenta and all—and thus form a lithopedion, which is also demonstrable roentgenographically, but that is a different process. It is extremely rare to find a lithopedion in the human uterus, but it is more common in the lower animals (Williams, *loc. cit.*).

It is important to recognize this complication as delivery may be delayed or hindered by the fetus papyraceous. In two of our cases, labor was delayed by the dead fetus presenting with the presenting part of the live twin. In the other three there was no complication; in fact, in one case (Case 2) the obstetrician did not realize that the placental structures contained a fetus papyraceous. In some of the reported cases a false diagnosis of the presentation was due to the fetus papyraceous presenting before the living twin. The third and most important complication which may arise is the retention of the fetus and its membranes after delivery of the normal twin. The atrophic and fibrosed placenta in several instances was quite firmly adherent to the uterine wall and was separated from it with difficulty. Retention of these non-viable tissues favors, of course, postpartum sepsis.

The roentgenograms have been useful in determining the age of the fetus papyraceous. The fetus does not lose any appreciable amount of its calcium content, and since it usually attains a fetal age of three to five months before death, the ossification centers are well developed. The usual body measurements, except for the foot length, are unreliable, as the fetus is flattened and compressed. We have, therefore, depended upon the appearance of certain centers of ossification, as described by Hess (8), for determination of fetal age, in addition to the usual body measurements.

The other case diagnosed antepartum by roentgenograms was reported by Schinz (19). A translation of his report is roughly as follows:

"As a curiosity I will describe an observation of my own in regard to twins. In this case one of the twins was undeveloped as a fetus papyraceous, whereas the other twin was a fully developed child (Fig. 2237). The diagnosis was correctly made before delivery.

"The diagnosis was made possible by a number of centers of ossification which were lying in one line below the head of the normally developed fetus visible at the entrance of the pelvis. The diagnosis was confirmed at delivery. The presenting fetus papyraceous had to be delivered first, following which the second fetus was born spontaneously."[2]

[2] This case was also reported and the illustration copied by P. H. Schumacher in *Ergebnisse der Medizinischen Strahlenforschung*, VI, 294, 1933, in his article on "Die Roentgendiagnostik in der Gebürtshilfe." We are indebted to Dr. Isaac Gerber for calling our attention to this reference.

Our five cases, in brief, are as follows:

Case 1. Mrs. J. A. C., 29 years old, primipara, wife of a physician. Family history and past history unessential. Catamenia began at 16 years, never regular, only two or three times a year, lasting one week. Last menstrual period was Nov. 21, 1936 (date of first visit to obstetrician, Feb. 1, 1937). Physical examination was negative. The duration of pregnancy was estimated at two and one-half months, but the patient was definitely certain that pregnancy did not take place until after Christmas. The menstrual periods had been so irregular that dates based upon cessation of menses could not be relied upon. The size of the uterus seemed much bigger than it should be, dating from a pregnancy after Christmas. Successive examinations on Feb. 19, March 12, and April 1 all indicated a larger uterus than normally expected in a pregnancy of the stated duration. From April 22, on to the termination of labor on Sept. 25, the uterus was not abnormally large. There was no history of injury or illness to account for the death of one fetus. There had been no bleeding or bloody discharge during this period. The Wassermann test was negative.



Fig. 1. Case 1. Antepartum roentgenogram three months before delivery, showing well ossified fetus papyraceous on left side of pelvis below and lateral to the head of the viable normal fetus.



Fig. 2. Case 1. Roentgenogram of fetus papyraceous after delivery, showing the degree of ossification of the fetal skeleton. Estimated bone age, 16.5 weeks.

Roentgenograms were taken on June 28, 1937, by Dr. Richard Dresser at the Collis P. Huntington Memorial Hospital (Fig. 1). These demonstrated a well developed fetus in cephalic presentation and a small fetal skeleton lying on the left side of the pelvis below and lateral to the normal fetal head.

The normal baby, a girl, was delivered Sept. 25, 1937, by Dr. R. S. Titus, by a simple forceps with the head in sight, after a short labor. The fetus papyraceous was delivered with the placenta and membranes, without difficulty. There was no bleeding after delivery.

The pathologic report, by Dr. Fred Ripley, Boston Lying-in Hospital (S-37-909), described the fetus papyraceous and the completely infarcted placenta with two amnii and one chorion. The two placentæ

were fused for a distance of 13 cm., but the infarcted fibrosed placenta of the fetus papyraceous was only 4 cm. wide at its widest point and only 4 mm. in thickness. It had been implanted low, either marginal or a partial placenta previa, a possible cause of the fetal death. The umbilical cord was 12.5 cm. in length. The fetus measured 14.5 cm. in (crown-rump) length, indicating an age of 18 weeks. The pregnancy was interpreted as single ovum twins.



Fig. 3. Case 2. Roentgenogram of fetus papyraceous after delivery. Skeleton well ossified but somewhat macerated. Estimated bone age, 21 weeks.

Roentgen examination of the fetus (Fig. 2) showed well defined centers of ossification for the ilium and ischium, but none for the pubic bone. A study of the other centers of ossification and comparison with the tables reported by Hess (8) gave a bone age of from 16 to 17 weeks.

Case 2 (S-35-34). E. O., aged 38, Para V, was delivered Jan. 12, 1935, of a normal male baby and a macerated male fetus papyraceous. Delivery was uncomplicated. The fetus is the largest in this group, with a C. R. length of 190 mm. and a foot length of 40 mm., with a resultant estimated age of 22 weeks. Dr. Hertig interpreted the placentæ and membranes as a double ovum twin pregnancy.

The roentgenogram (Fig. 3) shows the skeleton well developed with some collapse of the cranial bones. The age by development of the ossification centers is between 21 and 24 weeks, probably nearer 21 weeks as the center for the sternum is not present.



Fig. 4. Case 3. Roentgenogram of fetus papyraceous after delivery. Fetal skeleton poorly ossified, probably a case of osteogenesis imperfecta. Impossible to identify many centers of ossification. Bone age not determinable from roentgenogram.

Case 3 (S-35-204). Mrs. B., aged 23, went through a normal pregnancy, with no bleeding and no excess size of the uterus noted. She was delivered March 10, 1935, by Dr. Benjamin Tenney, with simple low forceps. The living twin was a full term male and the fetus also was male. It measured 110 mm. in C. R. length, 17.5 mm. foot length, which gave an estimated fetal age of fifteen and one-half weeks. There were two placentæ, the one

attached to the fetus papyraceous being shrunken, fibrosed, and immature.

The roentgenogram (Fig. 4) of the fetus shows ill defined bones of amorphous structure with multiple fractures and

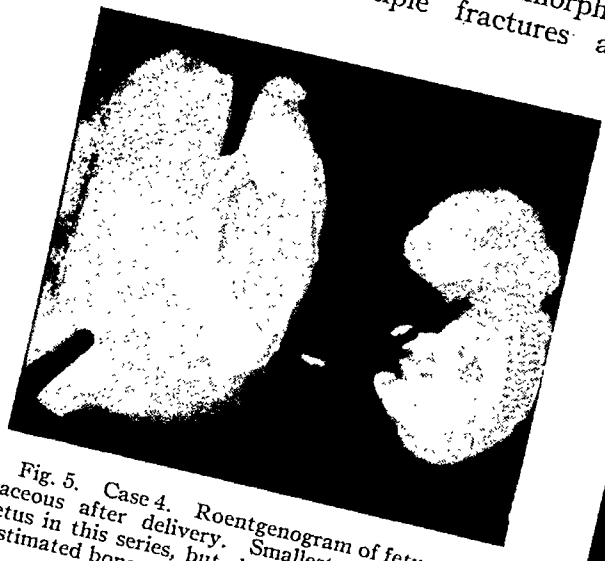


Fig. 5. Case 4. Roentgenogram of fetus papyraceous after delivery. Smallest and youngest fetus in this series, but skeleton is quite definite. Estimated bone age, 11 weeks.

abnormal ossification. This is presumably a case of osteogenesis imperfecta, in bivular twins. The bone age by roentgenograms was not determinable due to the poor detail and amorphous structure of the bones.

Case 4 (S-36-734). M. R., aged 44, wife of a WPA worker, was a pre-eclamptic, Grade 1, with moderate hypertension, in her tenth pregnancy. There was no history of injury or illness to account for the death of one twin. She was delivered at the Boston Lying-in Hospital on July 29, 1936, of a normal male baby by breech extraction, followed by the fetus papyraceous and placenta. The C. R. length of the fetus was 70 mm. (possibly inaccurate due to distortion). The foot length was 14 mm., estimated age 13 weeks. Dr. Hertig interpreted the pregnancy as biovular twins. The sex of the fetus compressus was not definite.

The roentgenogram (Fig. 5) of the fetus shows it to be the least developed of the five. The ossification centers indicate an age of only 11 weeks.

Case 5 (S-37-843). This patient, R. R., was delivered at the Boston Lying-in Hospital shortly before Case 1 was admitted. The patient, a primipara, aged 35, pre-eclamptic, Grade 1, was delivered



Fig. 6. Case 5. Roentgenogram of fetus papyraceous after delivery. Ossification centers indicate a fetal age of 14 weeks.

Sept. 10, 1937. Her last menstrual period was Dec. 8, 1936. Her past history revealed toxemia and mild hypertension, B. P. 170/98. Hinton and Wassermann tests were negative. She was delivered by low forceps after median episiotomy due to dystocia incident to secondary uterine inertia. The presence of the fetus papyraceous was not discovered until the secundines were examined in the department of pathology. The obstetrician noted a foul odor and "old calcified blood clot" when the placenta and membranes were delivered. The normal living twin was a female. The fetus (male) and placentæ weighed 620 grams. The umbilical cord of the compressed fetus was 53 cm. in length, and was looped once around the neck and twice around one arm. There were separate and intact membranes (amnion and chorion) around the fetus. The C. R. length of the fetus was 10.5 cm., sug-

gesting a fetal age of 15 weeks. The placenta of the fetus papyraceous was light yellow in color, firm, obviously infarcted and fibrosed. Figure 6 shows the roentgenogram of this fetus. The age by ossification centers was 14 weeks. The pregnancy was double ovum twins.

CONCLUSIONS

1. Fetus papyraceous is probably more frequent than the literature would indicate.

2. The diagnosis can reliably be made by antepartum roentgenograms.

3. The age of the fetus can be determined by the centers of ossification and it may be more reliable than the conventional measurements due to shrinkage or elongation by compression.

4. A case of fetus papyraceous, successfully diagnosed by roentgenograms three months before delivery, is reported, and the roentgenograms of four other fetus papyracii are included to demonstrate the ossification of the fetal skeleton in similar cases.

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THE ROENTGENOLOGIC ASPECTS OF BRONCHOMYCOSIS[1]

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ACCURATE roentgenologic diagnosis of pulmonary infiltrative lesions is notoriously difficult in many of the patients seen in our everyday work. In some of these cases we must resort to laboratory examination of the sputum in order to establish the diagnosis. This is especially true of the bronchomycoses in which the roentgen manifestations are so protean as to defy exact classification in most instances.

Pulmonary tuberculosis is the disease most often confused with the mycoses and many cases of bronchomycosis have been discovered in tuberculosis sanatoria after having been there for varying periods of time. Norris (7) reports the discovery of yeasts in the sputum of 15 per cent of the cases in a tuberculosis sanatorium. While undoubtedly these did not all represent cases of bronchomycosis *per se*, yet as secondary invaders they are of considerable importance in cases of chronic lung infections. Many cases of bronchomycosis are clinically and pathologically similar to tuberculosis and the two can be differentiated only by prolonged sputum examinations. It should be emphasized that the presence of fungi in the sputum does not in itself justify the diagnosis of a primary fungus infection but it should indicate further studies to determine what rôle they play in the patient's problem.

The incidence may show a geographical distribution. Coccidioidal granuloma was first described in California and it is largely confined to that portion of the country. Blastomycosis is more common in the central and southern States, and all mycoses are more frequently seen in the tropics.

A simple classification of the bronchomycoses is given by Castellani (3), as follows: 1. Due to yeast-like fungi, *viz.*, fungi of the types *Monilia*, *Cryptococcus*, *Saccharomyces*, *Blastomycoides*, and *Endomyces*. 2. Due to filamentous fungi: (*a*) of the slender type, *viz.*, fungi of the types *Nocardia*, *Anæromyces*, and *Vibriothrix;* (*b*) of the larger type, *viz.*, fungi of the types *Oidium* and *Hemispora;* (*c*) with characteristic fructifications—*Aspergillus*, *Penicillium*, *Mucor*, *Rhizomucor*, *Acremoniella*, *Sporotrichum*, *Acladium*, etc.

It is impossible in this communication to attempt the description of the changes produced by all of these organisms but a brief discussion of some of those more commonly encountered will be given.

Blastomycosis.—This is a highly fatal, uncommon disease which is caused by infection by blastomycetes—a variety of yeast-like fungi. Some believe that other organisms may be concerned in the etiology, but in many cases the organisms in pure culture have been obtained and the disease reproduced in animals.

The most characteristic lesion is a chronic skin lesion with abscess formation, ulceration, and thickening of the epidermis. These abscesses may involve the subcutaneous tissues, lymph nodes, viscera, bones, etc. Rupture of the abscesses, with discharging fistulæ, frequently takes place.

The pulmonary lesions are less common than those mentioned above, but a number of cases with postmortem examinations have been reported, so that the pathologic changes have been well established. Stober (11), after studying a number of these cases by postmortem examination, states that acute laryngitis and bronchitis are usually associated with active pulmonary lesions and pleural in-

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Fig. 1, Case 1.



Fig. 2, Case 2.

volvement. The active pulmonary lesions showed a great deal of fibrous tissue around areas of infected lung tissue, frequently in the upper lobes. A number of cases showed evidence of diffuse hematogenous infection.

The clinical picture often simulates that of tuberculous infection and, indeed, the pathologic picture at autopsy may at first be thought to be that of tuberculosis. To make a positive diagnosis, the presence of blastomycetes must be demonstrated in the lesions themselves, although if they are present in the sputum of a case in which the presence of tuberculosis can be excluded the diagnosis is highly probable.

The pathologic changes disclosed on the roentgenogram often simulate those seen in cases of tuberculosis. There may be considerable increase in the hilar areas and in the bronchovascular markings, with an unusual studding following these markings. Brooksher (1) quotes Dunham regarding a characteristic studding which follows one or more of the main trunks, but which does not quite reach the periphery. There is usually an area of lung density around the periphery.

In the same case there may be patches of bronchopneumonia. Some of the changes may suggest miliary tuberculosis but there is usually more fibrosis present in bronchomycosis. Pleural reaction is common in these cases. Cavities filled with necrotic material are of frequent occurrence.

Case 1. White male, aged 60. The patient was admitted to the hospital complaining of "lumps all over the body." For three months previous to the admission he had been expectorating a considerable amount of thick sputum, which was sometimes tinged with blood. A slight cough was also present. Three weeks previously a small lump appeared on the forehead; this was not tender or painful. Since then similar lesions had appeared all over the body, even in the roof of the mouth. These lesions were small, elevated, ulcerating areas with necrotic centers, from which oozed a seropurulent discharge. There was generalized lymphadenopathy.

Examination of the chest disclosed limited respiratory movement. There was impairment on percussion over both sides of the chest, and coarse moist râles over both upper lobes.

The roentgen films of the chest showed extensive parenchymatous infiltration throughout both lungs. The infiltration was of the miliary type with areas of coalescence in the bases, producing partial consolidation in those areas (Fig. 1).

A direct smear from a lesion on the left forearm showed typical blastomycetes. Biopsy of one of the lesions resulted in the finding of blastomycetes, which are seen as heavy rings with clear inner zones, usually lying in areas surrounded by round, wandering cells and polymorphonuclear leukocytes.

The patient declined rapidly and died on the sixth day after admission.

Postmortem examination: The significant findings were as follows: Widely scattered over the skin were fungoid lesions ranging from 1 to 4 mm. in diameter, and elevated above the skin as much as 1 cm. in places. The papillomatous mass was easily scraped away and consisted of a soft granular scab covering a slightly depressed shallow ulcer covered by thin, yellow, purulent material.

The lung margins were firm and rounded, similar to liver tissue. On sectioning the lungs, the smaller bronchioles were filled with purulent material. The pulmonary parenchyma was firm and densely infiltrated with small, gray, pin-point nodules which were quite firm in consistency. There were no areas of cavitation.

Similar areas of miliary infiltration were found in the spleen and kidneys. There were abscesses in the prostate gland and in both sternoclavicular joints.

Examination of material from all these areas showed evidence of cutaneous and systemic blastomycosis.

Actinomycosis.—This is an uncommon but not rare disease, caused by organisms with branching mycelia which often break into segments. The organism which is most commonly present is the bovine type or "ray fungus" and is recognized by the so-called sulphur granules found in pus. This disease is somewhat more common in those living in rural communities, and especially in those persons caring for cattle. It is not known, however, that the disease is acquired directly from cattle but probably comes from fungi which live on vegetation.

About 15 per cent of all the cases of actinomycosis observed clinically are found to involve the thorax. The infection may involve both the chest wall and lungs. Good (6) states that actinomycosis of the thorax falls into four groups: (1) broncho-actinomycosis; (2) pneumo-actinomycosis; (3) pleuropneumo-actinomycosis, and (4) thoracopulmonary actinomycosis, or those cases in which the thoracic wall is involved as well as the lungs.

Pulmonary infection is often secondary to infection in other parts of the body. Aspiration is another source of infection. This has been demonstrated by the finding of aspirated material in the lungs surrounded by the organism. The pulmonary pathology has been described by Christison and Warwick (4), according to the type of lesion present in the lungs. In the bronchitic type, pus and fungi are found; this type is rare. In the pneumonic type, the alveoli are filled with pus and it resembles a bronchopneumonia. These areas may coalesce and form abscesses. In the third type the abscesses may burrow through and involve the pleura where pus accumulates. It may then pass into the fourth type, in which the chest wall is involved. The lower lobes are usually involved first.

The symptoms are similar to those of other pulmonary infections, and include chills and fever of an irregular type, night sweats, loss of weight, pain, and abundant sputum which may contain pus or blood. The diagnosis is usually made by the finding of sulphur granules in the sputum.

The roentgen picture is not characteristic. Areas of consolidation may be present and in some instances there are localized nodules, as noted in the case report below. In those cases in which



Fig. 3, Case 3.

pleural involvement is present, there may be fluid in the pleural cavity. Sometimes cavitation in the form of abscess cavities are seen.

The disease must be differentiated from tuberculosis, but in some cases it may simulate lung abscess, tumor, or empyema.

In practically all cases with pulmonary involvement, the disease ends in the death of the patient. Potassium iodide in large doses seems to be of considerable benefit. Surgical excision of localized lesions has been curative but this is scarcely applicable to pulmonary lesions. Roentgen therapy to such lesions, combined with potassium iodide and possibly vaccine, would seem to offer the greatest hope of success.

Case 2. White male, aged 17. The patient was admitted with a complaint of draining sinuses in the abdomen. He stated that he had been well until 11 months previously, at which time he suffered a ruptured appendix for which he had a subsequent operation. Six months later, intestinal obstruction developed, for the relief of which several operations had been performed. Three weeks previously an abscess in the left lower quadrant was opened; several of these openings were draining at the time of the examination. There was loss of strength and weight, and his appetite was poor. The temperature was only slightly elevated.

Physical examination revealed evidence of several surgical incisions with draining sinuses over the abdomen, in which several masses were present. The lungs were essentially clear.

Smears of pus from the draining sinuses showed evidence of actinomycosis.

Roentgen films of the chest disclosed a large, smooth, rounded shadow of increased density in the base of the right lung just above the diaphragm. In view of the abdominal actinomycosis, it was felt that this lesion had the same etiology (Fig. 2).

Roentgen therapy was instituted and carried out over a period of several months. This produced a remarkable improvement in the patient's general condition. He gained weight and strength, and his color and expression were considerably improved. The palpable abdominal masses almost disappeared. X-ray films of the chest at that time showed a diminution in size of the mass in the base of the right lung but not disappearance of it. The patient then left and we have been unable to trace him farther.

Moniliasis.—Attention was first directed to this condition by Castellani (3), in Ceylon, in 1905. It is now conceded that while *Monilia* may be the cause of primary bronchopulmonary disease, it may also be present as a secondary invader in cases of primary bacterial infection. *Monilia* is usually a saprophyte and is frequently found on decayed vegetable matter. In some cases it appears to be a harmless invader; in others it becomes pathogenic to either man or animal.

Two types of clinical cases are described by Castellani (3): a mild type and a severe type. In the mild type the patient exhibits few symptoms or signs. He may be ill a few weeks or months and clear up spontaneously, or the disease may continue into the severe type.

In the severe type the history suggests tuberculosis. There is weight loss, fatigue, fever, cough, and sometimes hemorrhage.

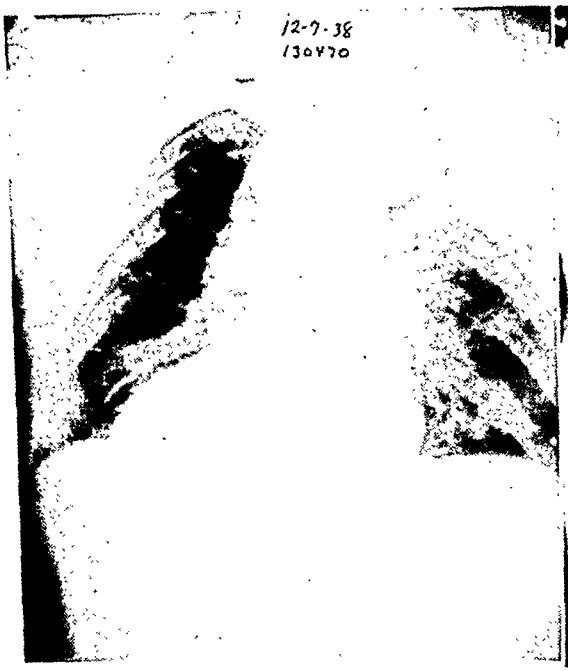


Fig. 4, Case 4.

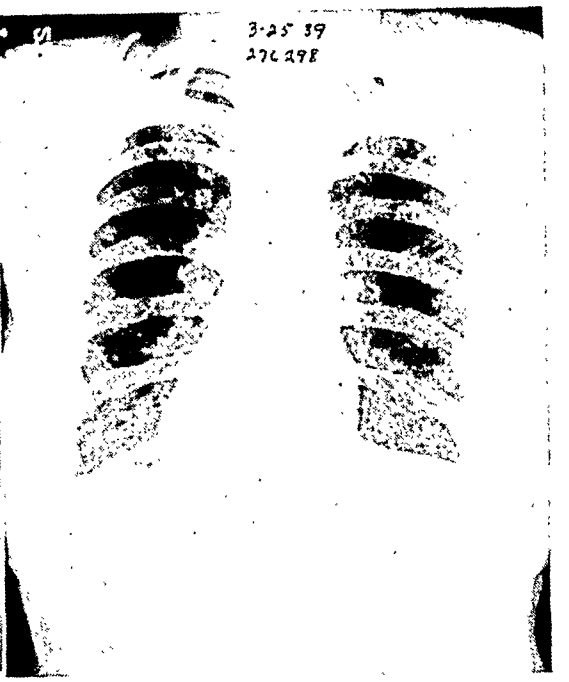


Fig. 5, Case 2.

Physical examination may reveal evidence of pleural thickening and sometimes consolidation. The apices of the lungs tend to be clear. Chronic cases may extend over a period of years, with omissions, but the acute cases often progress rapidly to a fatal termination.

The diagnosis of pulmonary moniliasis rests upon the constant presence of monilias in the sputum and the absence of tubercle bacilli. The sputum must also be collected with every precaution to avoid external contamination.

Roentgenograms of the chest show a variety of changes. There is usually a marked enlargement of the hilar areas, with extensive infiltration extending from these areas throughout the lungs. In some cases there is a patchy type of infiltration present in various parts of the lungs. This differs somewhat from tuberculous infiltration and is usually less marked in the pulmonary apices. There is generally an extensive fibrosis forming a network of infiltration throughout the lungs. There may be considerable studding along the bronchovascular markings. We had a series of five patients, all in one family, in which miliary changes were present throughout both lungs. These were somewhat coarser than the nodules of miliary tuberculosis but otherwise were similar. No tubercle bacilli were ever found in these cases (Fig. 5).

Postmortem examination usually shows replacement of the pulmonary parenchyma by a diffuse fibrosis. The bronchi may contain exudate which has many round and wandering cells. The actual monilia are usually present in large numbers. There may be small abscess cavities about the smaller bronchioles and these contain monilia.

Castellani (3) suggests the use of potassium iodide with which glycerophosphates and balsamics may be associated. He calls attention to the fact, however, that in certain instances potassium iodide is practically without beneficial effect. Vaccines may be helpful in some cases.

The following report is of a case of ours which came to the postmortem table.

Case 3. White male, aged 69. The patient came to the hospital complaining of weakness, cough, shortness of breath, and loss of weight. The symptoms were

first noticed six months previously, beginning with a non-productive cough, which later became productive of a white foamy sputum.

Physical examination disclosed an emaciated man 50 pounds underweight, coughing and expectorating. There was impairment on percussion over the upper chest. Bronchial breathing and showers of fine and medium moist râles were heard throughout both lungs. There was clubbing of the fingers.

The sputum was repeatedly negative for tubercle bacilli, and the tuberculin test was negative. The sputum culture showed the presence of *Monilia* repeatedly.

Roentgenograms of the chest disclosed evidence of hilar enlargement on both sides and an extensive network of fibrosis throughout both lungs. Some nodular development was present in both lungs. There was slight pleural reaction along the axillæ. The upper dorsal spine showed a mild degree of scoliosis and the trachea was displaced to the right side (Fig. 3).

Potassium iodide was administered but had no appreciable effect on the course of his illness. Death occurred three weeks after admission.

At the postmortem examination the following significant changes were found. The pleural surfaces were covered with a fine fibrous deposit, less marked over the apices. The hilar nodes were enlarged and on section were soft and deeply pigmented. The large bronchi contained a moderate amount of stringy, almost gelatinous, mucopurulent material. There was greatly increased density of the pulmonary parenchyma. The cut surface had a firm fibrous appearance and a mottled grayish-white color. There were multiple small areas of bronchiolar dilatation, scattered through the lungs and other small areas which suggested patches of bronchopneumonia. The consolidated process had somewhat the appearance of a caseous pneumonia but was firmer in consistency, dryer, and more fibrous in character. Bacterial examination of the sputum disclosed *Monilia*. Microscopic examination showed almost complete replacement of the pulmonary parenchyma by diffuse fibrosis.

Aspergillosis.—This disease is the result of infection by one of the *Aspergillus* fungi. It is frequently present in pigeons, and in some countries pigeon-breeders acquire the disease. It is not common in this country as a primary pulmonary disease but has been noted occasionally as a secondary invader in the course of other diseases.

Schneider (9) states that the onset of the infection is insidious. The symptoms are much the same as in tuberculous infection. There is often a productive cough with frothy purulent sputum. Hemoptyses are frequent. Schneider says that patients with this disease have a rather healthy appearance and do not look ill or emaciated, as they often do in tuberculosis and other chronic lung diseases.

The roentgen films of cases of primary pulmonary aspergillosis reported in the literature, have not shown any characteristic findings. There is usually evidence of fibrosis and emphysema, together with some pleural reaction. The picture is usually mistaken for that of tuberculosis.

Other filamentous fungi having characteristic fructifications which are of clinical importance are *Penicillium* and *Mucor*. These are often saprophytes and not primary invaders. They are often present on decayed vegetable matter and moldy hay. Fawcitt (5) has described numerous cases of workers with moldy hay, who showed severe clinical symptoms. He applied the name "bronchomycosis feniseciorum" to this condition. The first symptoms include increasing shortness of breath, cough with frothy sputum, and slight fever. The sputum usually contained a mixed infection of *Aspergillus*, *Penicillium*, and *Mucor*. He described the roentgen changes in four stages beginning with mild peribronchial changes, passing into mottling that may be mistaken for miliary tuberculosis. There is then enlargement of the hilar areas. Later there are patches

of increased density due to coalescent areas of fibrosis. There is restriction of motion of the diaphragm, emphysema, and cardiac enlargement.

This infection, he believes, may provide a soil for the development of such diseases as tuberculosis or malignant disease. Sayers and Meriwether (8) reported the discovery of fungi in the sputum of 31 cases of miliary lung disease with calcification. They were present in every case examined and consisted of several types of *Aspergillus* fungi. We have had one case of *Penicillium* infection which was probably not the primary cause of death.

Case 4. White male, aged 69. The patient was admitted complaining that five days previously he had become ill with chills and fever. There was a moderate cough which was productive of thick, grayish-white, mucoid material. The sputum was occasionally blood-tinged, but there was no frank hemoptysis.

Physical examination of the chest disclosed evidence of impairment over the chest posteriorly. Breath sounds were distant over the impaired areas, but there were no tubular breath sounds. Many yeast cells were found in the sputum. It was felt that this was an atypical case of pneumonia. *Pneumococcus*, Type VIII, was cultured from the blood.

Roentgen films of the chest showed evidence of extensive infiltration throughout the right lung, especially the right apex, with small areas of localized hazing suggesting bronchopneumonia. Fungous infection was suggested as a possibility (Fig. 4).

During the next few days the clinical condition of the patient became worse and the roentgen films showed progressive involvement of the lungs. He died on the fifth day of hospitalization.

Postmortem examination disclosed evidence of bilateral bronchopneumonia; bilateral acute bronchitis; fibrous pleuritis; calcified and caseous lymph nodes, right, and various lesions in other parts of the body. Cultures from the lungs resulted in the finding of *Penicillium*.

It was our feeling that the *Penicillium* infection was not the cause of death.

Coccidioidal Granuloma.—The infectious agent in this disease is the *Oidium coccidioides* which produces a generalized disease which may attack many of the bodily structures. It more commonly involves the skin, subcutaneous structures, glands, osseous system, and pulmonary structures. It is frequently primary in the pulmonary structures.

Nearly all of the cases have been reported from areas in this country west of the Rocky Mountains, but a few have been found in other parts of this country and South America. Most of the reported cases have been from California; we have no records of this disease in our files. Carter (2) has written an excellent report on this condition, deserving study by those interested in the subject, in which he describes the pulmonary lesions of this disease as consisting of extensive miliary lesions, nodular consolidations with caseation, massive consolidation, and abscess formation. By roentgen examination he found that approximately half of his cases, with pulmonary lesions, showed the miliary type of involvement. These miliary lesions appeared somewhat more obscure on the film than in corresponding cases of miliary tuberculosis. Considerable hilar enlargement was present in about 80 per cent of the cases; a mediastinal mass in about one-half. Some type of consolidation was shown by about 90 per cent of the patients reported. Little evidence of fibrosis could be seen from the films. A few of the cases showed evidence of pleural involvement, and several small cavities were seen in the upper lobes.

Treatment of these patients is far from satisfactory as no specific medication is available. Most of those with generalized infection suffer severe toxic prostration and death. The lesions resemble those of blastomycosis and tuberculosis.

Streptothricosis.—This is a relatively rare infection in man. Castellani (3) states that it is one of the most serious types of bronchomycosis, and usually ends

in death. There is weight loss, anemia, and sputum which is first mucopurulent and later blood-tinged. The physical examination may be negative or suggest scattered patches of infiltration in the lungs.

Singer and Ballon (10) have reported a case in detail, with a review of the literature. These authors believe the primary infection was in the lungs, but the fungus was found at autopsy in various parts of the body. There was fever, leukocytosis, and symptoms referable to the various affected organs. The lesions in the lungs showed extensive nodular infiltration similar to that seen in miliary tuberculosis. There was also evidence of necrosis and abscess formation. Tuberculosis was also present in the lungs, associated with it, and tubercle bacilli were demonstrated in direct smears from pus obtained from the abscess cavities, but had not been demonstrated in the sputum during life. In the differential diagnosis of streptothrix infection of the lungs, these investigators state that one must consider tuberculosis, actinomycosis, sporotrichosis, and glanders; also pyogenic lung infections.

We have had no streptothrix infections of the lungs.

DISCUSSION

It is self-evident from the above summary of the various bronchomycoses that there are no characteristic roentgen changes that will enable us clearly to differentiate these diseases from each other or from other diseases. There are, however, certain suggestions and also positive findings which may be obtained from a study of the roentgen films which will materially aid in arriving at a differential diagnosis.

The roentgen findings in all these bronchomycoses simulate, to some degree, pulmonary tuberculosis or some of its complications. In certain cases, bronchomycosis is suspected only because tubercle bacilli cannot be demonstrated after prolonged search for them, and because the clinical course of the disease is not typical of tuberculosis. Demonstration of the fungus will then lead to a correct diagnosis. Associated extrapulmonary lesions, such as skeletal lesions, fistulæ, and granulomatous skin lesions, are also an aid in directing attention to the search for fungi.

Some of the roentgen findings and their occurrence in the various bronchomycoses are miliary nodules. These are present in some form or other in all these diseases, except actinomycosis and aspergillosis. In the case of the latter, when it is combined with *Penicillium* and *Mucor*, as in hay workers, this is a prominent feature of the roentgen picture. In blastomycosis there is usually more fibrosis than in miliary tuberculosis. In certain cases of moniliasis there is studding along the bronchi and nodules which are usually larger and less discrete than those of tuberculosis (Fig. 5). Miliary nodules are also quite common in coccidioidal granuloma and streptothricosis.

Pulmonary necrosis with abscess formation is seen at some stage of the disease in a number of these infections. In blastomycosis, abscess formation is sometimes found, although in the case reported above this was not present. This complication is quite common in cases of actinomycosis and may secondarily involve the chest wall with the formation of persistent fistulæ. Small cavities may be present in moniliasis and coccidiosis but it is not a prominent feature of these diseases. Singer and Ballon (10) report abscess formation in their case of streptothricosis.

In many of these cases the occurrence of pulmonary fibrosis is the prominent feature of the lesion. It is well marked in the lesions of blastomycosis and may extend far up into the apices. In moniliasis there is usually a replacement of the lung parenchyma by a diffuse fibrosis. Fibrosis with an accompanying emphysema is seen in aspergillosis and penicillosis. It is present to some extent in the other bronchomycoses but it is not as prominent a feature of these diseases.

Pleuritic involvement, with or without

empyema, may be present in any of these infections and is frequently an outstanding feature of actinomycosis with secondary involvement of the chest wall. In nearly all cases reported with postmortem findings, there was evidence of pleural changes.

Areas of a patchy type of consolidation, similar to bronchopneumonia, are also present at some stage of the disease in most of these infections. Sometimes these lesions appear to represent areas of primary involvement which may coalesce and break down with the formation of abscesses. In other instances they appear to be due to a terminal bronchopneumonia which is often the direct cause of death.

Sometimes basilar involvement is more common than the apical involvement, which may be of value in suspecting nontuberculous infection. There is often considerable hilar enlargement and in coccidiosis there was a mediastinal mass present in almost half of Carter's (2) cases.

SUMMARY

The bronchomycoses, while not rare, are not discovered with great frequency, and until the past few decades comparatively little attention was devoted to them.

The symptoms and signs are similar to those of tuberculosis and often they are at first thought to be tuberculous in origin. Whenever the history or x-ray changes are atypical of tuberculosis, the sputum should be examined microscopically and also cultured repeatedly, so that these cases may not be treated as tuberculous cases.

In early stages, these patients appear to have a mild bronchitis with some expectoration; later they develop many of the signs and symptoms and even the complications of tuberculosis. The final diagnosis rests on the discovery and identification of the offending organism.

The roentgen changes are suggestive of a differential diagnosis in some instances but usually one can only suggest the presence of a fungus infection. We have attempted to describe some of the more prominent roentgen signs of the various bronchomycoses.

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DIFFERENTIAL DIAGNOSIS OF NON-SPECIFIC INTERSTITIAL PNEUMONITIS[1]

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THE terms "pneumonitis" and "pneumonia" are synonymous; both may be defined as "inflammatory disease of lung parenchyma." Many pathologists, however, reserve "pneumonitis" for use in designating pneumonia of the chronic interstitial fibroid type, which may be encountered under a wide variety of clinical circumstances. As a result, "pneumonitis" has acquired an implied limitation of meaning.

Although lobar pneumonia in its classical form is, strictly speaking, a form of pneumonitis, the distinctive clinical and radiologic features of this disease justify its specific designation as lobar pneumonia by the radiologist. Likewise, in the majority of instances pulmonary tuberculosis can be identified radiologically with a reasonable measure of confidence. It is only when the telltale signs characteristic of some particular pathologic process are lacking that the radiologist feels the desire to avail himself of the latitude which the widely inclusive term of "pneumonitis" implies.

Chronic inflammatory processes in the lungs are prone to result in fibrous scarring within the alveolar walls and about the bronchi. This state of affairs can be demonstrated microscopically in a number of situations, such as chronic fibroid tuberculosis, silicosis, and unresolved lobar pneumonia. Since it is by no means possible to differentiate the various disease entities which may be associated with this type of pulmonary inflammation on the basis of roentgen findings alone, it is both sensible and convenient for the radiologist to report findings which suggest chronic fibroid pneumonia as "interstitial pneumonitis," making no attempt to identify the etiologic factor unless circumstantial evidence of some sort is available to identify the causative agent. Because the various inflammatory processes which may affect the lungs are so variable in their radiologic expression it is extremely difficult, if not frankly impossible, to describe in terms of location, configuration, size, and relative density the various appearances which might be considered characteristic of non-specific interstitial pneumonitis.

If one were to use a process of exclusion, lobar pneumonia might be set apart because, if seen in relatively early stages, this disease produces almost uniform abnormal opacity of the lungs, sharply limited in at least a part of its periphery by the pleural surfaces of the involved lobe or lobes. The pneumonic lung under these circumstances frequently shows radiographic evidence of slight shrinkage. Periodic examination at relatively short intervals is very enlightening because the radiologic manifestations of the disease notoriously lag behind the clinical signs, and the entire process resolves and disappears in short order unless complications develop.

Lobular or bronchopneumonia presents a different radiologic appearance characterized by patchy abnormal density becoming confluent if the zone of involvement is sufficiently large. This form of pneumonia may be more widespread than is customary in the lobar type, though it is not uncommon to find the disease confined to a single lobe at any one time. Either of these types may involve multiple lobes or may progress from one part of the lung to another. All forms of pneumonia can be produced by a variety of organisms. As an example of one of the less common etiologic factors,

[1] Read before the Twenty-fifth Annual Meeting of the Radiological Society of North America, at Atlanta, Dec. 11-15, 1939.

the fusiform bacillus and the spirochete of Vincent may be cited. There is little in the radiologic appearance of pneumonias, in general, to indicate the identity of the causative agent. However, the time factor involved in the process of resolution as determined by repeated roentgen examination is sometimes of considerable assistance.

The aspiration of foreign substances into the air passages constitutes an insult to the lung which is commonly associated with inflammatory reaction. Aspiration pneumonia is not infrequent following general anesthesia and even after local anesthesia of the upper respiratory tract. Derangement of the nervous mechanism controlling the epiglottis and the muscles of deglutition may permit ingested materials to be aspirated into the chest with the resultant development of pneumonia. To cite a case in point, a zone of pulmonary inflammation was recognized deep in the right lower lobe of a patient subsequently shown to have an annular neoplasm of the esophagus producing relatively high grade obstruction and consequent spill-over of esophageal contents into the trachea and bronchi. The process had become chronic because of often repeated insult to the same portion of lung. The pulmonary lesion was not associated with the usual abrupt and alarming clinical manifestations of pneumonia.

Following the aspiration of solitary foreign bodies and even without the blockage of air supply to large areas of lung, the development of local inflammatory disease is commonplace. So long as the foreign body remains, inflammatory disease persists, ultimately becoming chronic with the production of non-reversible scarring.

Sometimes the resolution of acute pneumonia is incomplete. Reparative processes are interrupted by recrudescence and active suppuration may occur locally with liquefaction necrosis and abscess formation. Before an abscess cavity can be demonstrated radiologically, before evacuation of necrotic material has occurred, lesions of this sort are exceedingly difficult to identify, with certainty, on the basis of a single radiologic observation.

One of the most commonly encountered pulmonary situations, in which characteristic radiologic evidence can be obtained, is

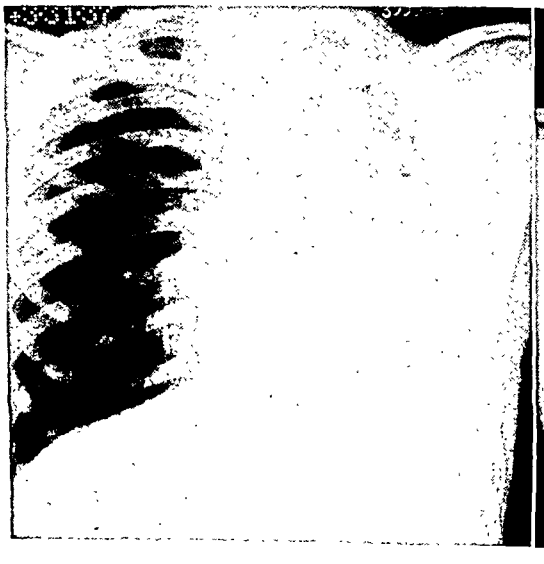


Fig. 1-*A*.

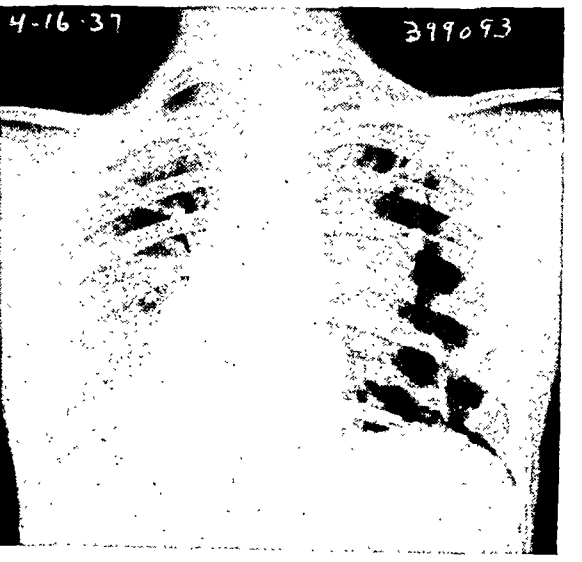


Fig. 1-*B*.

Fig. 1-*A*. Colored female, aged 15 years. Atelectasis, mediastinal displacement, multiple cavities in the lung. Far advanced pulmonary tuberculosis with multiple cavities and scar-tissue contracture suspected.
Fig. 1-*B*. Same case following diagnostic pneumothorax, showing absence of extensive intrapleural adhesions. Multiple cavities in the lung. Ultimate diagnosis: obstruction of left bronchus by adenoma producing profound bronchiectasis and interstitial pneumonitis.

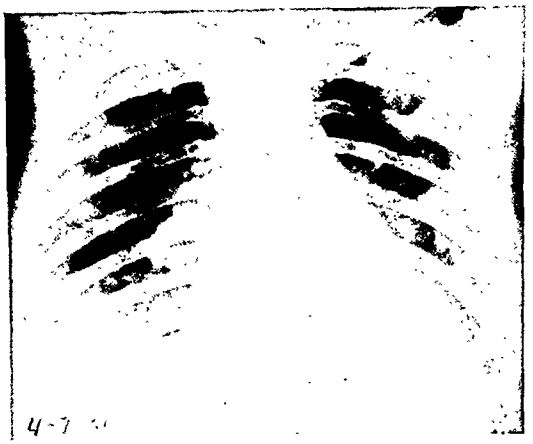


Fig. 2. Patchy pneumonitis representing actinomycotic pneumonia, confirmed at autopsy.

bronchiectasis. There is reason to believe that in a majority of these cases chronic interstitial pneumonitis precedes and, in fact, is instrumental in producing bronchial ectasia. Rarely, microscopic examination yields evidence of primary or congenital bronchial defects with superimposed inflammatory disease. One case in point might be mentioned in which, upon removal of the diseased lobe, the pathologist was able to demonstrate the widespread absence of certain elements in the bronchial wall. The existence of bronchiectasis may also be confidently suspected on the basis of radiologic evidence of interstitial pneumonitis corresponding in distribution to one or more of the major bronchi. It is not always possible to exclude the existence of bronchiectasis without recourse to bronchography. This is notably true even in the case of individuals in whom bronchiectasis is known to exist in the lower lobe of one lung, for example; for it is by no means an infrequent experience to find less extensive bronchiectasis in portions of another lobe, showing no signs of pneumonitis.

Pneumonoconiosis, whatever the irritative agent, presents, sooner or later, the microscopic appearance of chronic interstitial fibroid pneumonia or pneumonitis with characteristic whorl-like deposits of fibrous tissue. Striations within these whorls are usually to be seen in contrast to the hyalin changes which are common in tuberculous pulmonary fibrosis. Radiologically, the late stages of pneumonoconiosis are quite characteristic, rendered so by the tendency of multiple punctate opacities in the lung to coalesce at the level of the carina. Before this stage is reached, however, the lesions of silicosis can be closely imitated by a number of diseases of far different nature. It is well to remember that the radiologic signs which characterize this disease are produced by chronic interstitial pneumonitis which differs but slightly from the same condition which may be produced by a number of other factors.

Following therapeutic irradiation directed to the chest and adjacent structures, there may develop within that portion of the lung subjected to relatively great intensities of radiation a series of pathologic changes which, from the microscopic viewpoint, are again to be described as interstitial pneumonitis. In roentgenograms, the lesion is at first seen as a relatively uniform shadow with poorly defined margins. With the passage of time, these characteristics are supplanted by radiating strand-like shadows following the general course of bronchi in the affected area. If the injury to the lung has not been excessive, complete disappearance of the radiologic manifestations may be expected. Not infrequently, however, residual scarring can be recognized as a prominent aftereffect.

With the foregoing considerations as a background, it can be recalled from the experience of every radiologist that unusual manifestations of commonly encountered diseases and confusing radiologic findings in rare and unsuspected situations must be expected when accurate differential diagnosis is attempted. In patients presenting the classical story of cardiac failure and exhibiting unequivocal clinical signs, pulmonary congestion and edema as a result of circulatory embarrassment are anticipated and, therefore, easily recognized. Occasionally, degrees of circulatory failure capable of producing visible alterations in

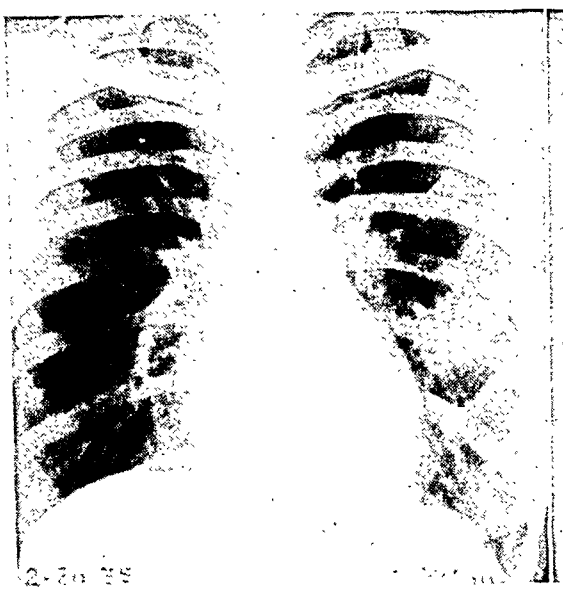


Fig. 3-*A*. Localized pneumonitis, left lung.

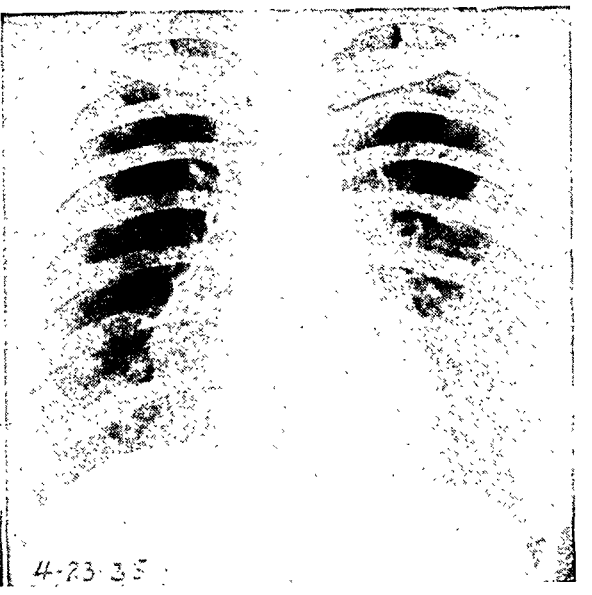


Fig. 3-*B*. Virtual complete disappearance following antisyphilitic therapy.

the appearance of the lungs are encountered by the radiologist, without warning. Under these circumstances, pulmonary edema and vascular congestion may be misinterpreted as evidence of inflammatory disease.

Long-standing bronchial obstruction with profound bronchiectasis of all lobes, produced by a small adenoma strategically placed in the left stem bronchus, produced in the chest roentgenograms of a 15-year-old negro girl, a very startling appearance. The heart shadow was lost in the generalized density throughout the left hemithorax and the trachea was sharply deviated to the same side. The contours of the diaphragm on the left and the left border of the heart could not be identified. In the upper portion of the chest and along the left axilla, definite signs of multiple cavities throughout the lung could be seen, leading to the initial impression of far advanced unilateral pulmonary tuberculosis with extensive cavitation and fibrous-tissue contracture. This impression was certainly strengthened by the age, sex, and race of the patient. Grid films of the chest, the entire absence of suggestive findings on the right, and the absence of confirmatory proof in the form of positive sputum analysis introduced doubt regarding the accuracy of diagnosis. Diagnostic pneumothorax proved the left lung to be entirely free from intrapleural adhesions, a situation scarcely to be expected in chronic fibrotic tuberculosis, and bronchography showed the imprint of the bronchial tumor in the column of iodized oil abruptly terminated in the left main-stem bronchus. After removal of the entire left lung, microscopic examination showed a profound degree of bronchiectasis involving all bronchi as well as far advanced, chronic, interstitial, fibroid pneumonia throughout the entire lung. This situation was scarcely to be expected on the basis of the initial chest roentgenograms (Fig. 1).

A patient desperately ill with high fever and definite clinical signs of widespread pulmonary disease presented radiologic findings difficult to explain. Irregular patches of abnormal density were to be found scattered throughout both lungs. These patches of abnormal density might have been considered as evidence of multiple pulmonary infarction or as widely separated zones of bronchopneumonia shortly followed by massive pleural effusion

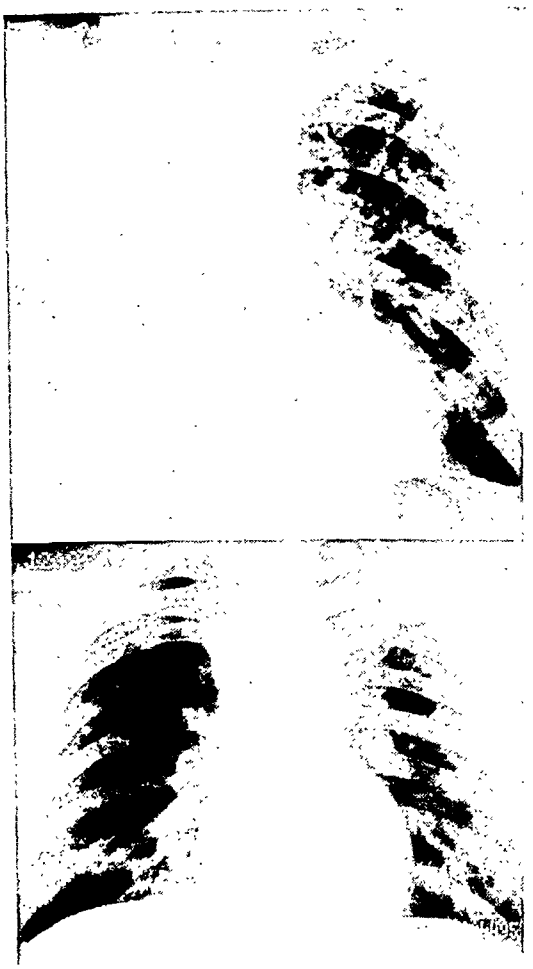


Fig. 4 (*above*). Bronchogenic carcinoma, upper lobe, right, with extension to other lobes of the same lung and pleura. Multiple metastases in the left lung.

Fig. 5 (*below*). Metastasis of squamous-cell carcinoma on dorsum of left hand to chest wall and lung, right apex. Radiation pneumonitis in axillary portion of the left upper lobe.

on the left. The patient continued on a rapid downhill course which was shortly fatal. At autopsy, widespread actinomycotic pneumonia was discovered and subjected to proof by morphologic and bacteriologic methods. In retrospect, the roentgenograms present no characteristics to warrant identification of the causative agent (Fig. 2).

In the case of a woman entering the hospital for the diagnosis and treatment of an open ulcerating lesion on the dorsum of one foot, radiologic examination of the chest—carried out as a means of weighing the likelihood of tuberculous etiology—showed a most unusual and highly localized though indistinctly outlined zone of increased density in mid-lung on the left. Certainly, this could not be considered characteristic of tuberculosis and was of necessity interpreted as "localized pneumonitis, etiology indeterminate." On dark-field examination a day later, spirochetes were identified in material taken from the ulcer on the foot and antisyphilitic therapy was at once begun. Closely paralleling progressive healing of the skin lesion, the abnormally dense patch of lung was observed to regain progressively its normal appearance, leaving no doubt as to its syphilitic nature (Fig. 3).

As a rule, pulmonary metastases from malignant neoplasms are seen in roentgenograms as sharply circumscribed, lobular densities buried in lung substance. This is not always the case, however, for rarely pulmonary metastases take the form of relatively small, flake-like densities of more or less uniform size which may easily be mistaken for evidences of silicosis, miliary tuberculosis, or other lesions of this sort. In one case in which this situation was encountered, the entire hemithorax on the right was largely obliterated as the result of a primary bronchogenic carcinoma in the upper lobe with direct extension and metastasis to the middle and lower lobes and the pleura, and with the further complication that the left lung was sprinkled with small granular metastases. Under these circumstances, far advanced pulmonary tuberculosis on the right, with subsequent miliary spread to the opposite lung, would certainly occur to most observers as the more likely explanation (Fig. 4). In another case under observation for a considerable period because of a large basal-cell carcinoma involving the dorsum of the hand, treated by amputation at the mid-forearm, re-amputation at a higher level with complete axillary dissection, and post-operative radiation, examination of the chest showed a smooth, hemispherical mass, close to the axilla high on the right, with underlying rib

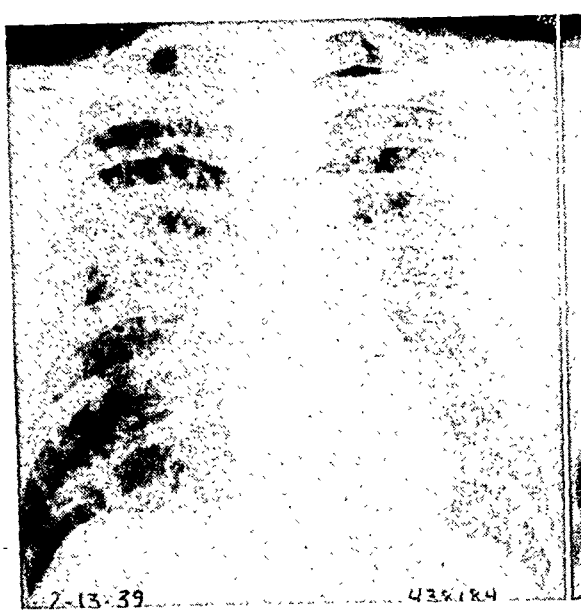


Fig. 6-*A*. Miliary type of pneumonitis in both lungs; diagnosis indeterminate.

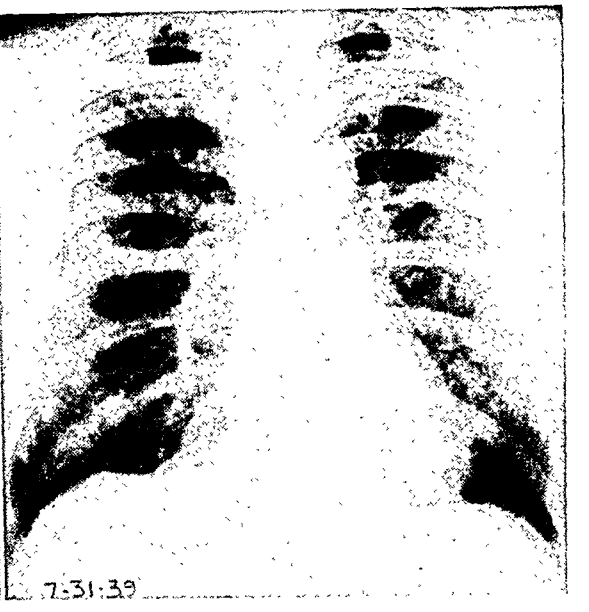


Fig. 6-*B*. Rapid resolution following treatment for coccidiomycosis.



Fig. 7-*A*. Patchy pneumonitis, representing sarcomatous type of Hodgkin's disease.

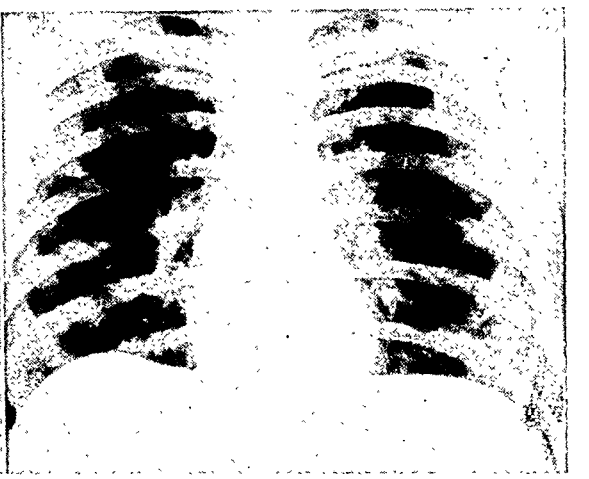


Fig. 7-*B*. Same chest following radiation therapy.

destruction, and a lesion in the left apex highly characteristic of moderately advanced pulmonary tuberculosis. As a matter of fact, the apical lesion on the left represented nothing more than radiation fibrosis to be expected as a result of the heavy irradiation employed in attempting to control this rapidly fatal and unusually metastasizing squamous-cell carcinoma (Fig. 5).

Another patient subjected to examination, because of continued temperature elevation and symptoms related to the chest, presented a striking and appalling appearance of extensive miliary tuberculosis. Repeated sputum examination and the persistent search for stainable tubercle bacilli when extended to include the consideration of other causative agents resulted in the conclusive diagnosis of coccidiosis, which responded nicely to treatment (Fig. 6). The appearance of this chest,



Fig. 8. Widespread bilateral pneumonitis and pleuritis. Leading occupational history. Diagnosis: pneumonoconiosis. Extensive Hodgkin's lesions in both lungs, hilum lymph nodes, spleen, and other organs were shown at autopsy. Also various stages of pulmonary tuberculosis and microscopic evidence of pneumonoconiosis.

when compared with that of another patient in which miliary tuberculosis was proved, provides no distinctive criteria upon which to base an accurate differential radiologic diagnosis.

The answer to the problem raised in another situation, in which peculiar splotches of opacity were observed scattered about throughout both lungs, was obtained as a result of microscopic examination of a palpable peripheral lymph node which showed unequivocal signs of Hodgkin's disease. By means of irradiation, the pulmonary lesions were caused to disappear quickly (Fig. 7). In another patient with a leading occupational history to indicate the probability of silicosis, this situation was diagnosed on the basis of punctate and coalescent opacities throughout both lungs with more pronounced and widespread pneumonic signs on the right. This patient shortly came to autopsy at which time Hodgkin's disease of an atypical form was clearly identified throughout both lungs, in bronchial lymph nodes, spleen, and numerous other localities throughout the body. In addition, there were definite evidences of pulmonary tuberculosis in various stages of the disease and in certain areas where the lymphoblastoma and the tuberculosis did not successfully obscure its characteristic signs, the typical arrangement of fibrous scarring seen in silicosis plus recognizable evidences of siderosis were observed and described by the pathologist (Fig. 8).

Summary.—Chronic interstitial pneumonitis is commonly encountered in radiologic examinations of the chest. The radiologic manifestations of this pathologic process may offer little evidence to suggest the particular causative agent. It is not at all uncommon that the etiologic factor may escape identification during the gross examination of pathologic material, and oftentimes the combined methods of physical diagnosis, radiology, pathology, bacteriology, and serology are required to identify the true nature of the disease. "Chronic non-specific interstitial pneumonitis" can be employed by the radiologist as a useful term to indicate that he has found what he believes to be evidence of long-standing inflammatory disease of the lung, the available characteristics of which will not permit more accurate classification.

THE MINIATURE X-RAY CHEST FILM[1]

By HOLLIS E. POTTER, M.D., *Chicago*, BRUCE H. DOUGLAS, M.D., *and* CARL C. BIRKELO, M.D., *Detroit, Michigan*

THERE have been many attempts to photograph the fluoroscopic image using both cinema and still camera methods, some of which have been to some extent successful. Growing out of the desire to provide a simple, inexpensive, and yet reasonably accurate method for the surveying of large numbers of individuals for the presence of tuberculosis or other diseases of the lungs, there have been developed several schemes for photographing the fluoroscopic image of the chest. De Abreu (1, 2), of Rio de Janeiro, should be given credit for having, in 1936, first applied such a method to surveys of large numbers, employing a contax camera for photography of the fluoroscopic image on 35 mm. film, the actual space used being 24 mm. These very small films can be economically taken but must, of course, be enlarged before they can be interpreted. Using this method, de Abreu and his associates have examined several hundred thousand persons and believe it to be practical for mass examination. They report being able to examine 1,000 persons per day with one machine. Janker (3) and Ulrici (4), as well as others in Germany have used this technic quite extensively and have reported favorably upon it.

Lindberg (5, 6), in this country, has adopted de Abreu's technic with some modifications of his own, and, while recognizing it as a cheap method for the examination of large numbers, in that it is more useful than the fluoroscope alone, believes that methods for the making of full size films should be improved upon rather than a definitely less accurate method.

Potter (7), a collaborator in this paper in consultation with the General Electric X-ray Corporation Laboratories, after much study and experimentation arrived at the conclusion that the most desirable objective in photography of the fluoroscopic image should be the production of a small film—small enough to constitute real economy in its use, and yet large enough to be readily interpreted without enlargement, or at least no greater enlargement than that provided by a simple reading glass. Working on this basis it was found that a 4×5 inch film with an effective focal range slightly smaller than this would answer the requirements. Special equipment was, therefore, designed for producing such photo-roentgenographs, and it is an evaluation of this method with which this study deals.

The technic of producing this film in our institution consists of the following factors: A modern x-ray installation with rotating anode tube capable of handling 400 ma. of current at from 60 to 80 kv., an impulse timer, and a tube–screen distance of 50 in. (or 125 cm.), thus producing as little distortion as possible. The time necessary for exposure depends on the thickness of the chest and varies from one-twentieth to four-twentieths of a second.

The fluorescent screen used is nearly seven times as fast as the usual Patterson screen. The lens is also a specially prepared instrument with a rating of F. 1.5 and is mounted in a tunnel with the screen at the opposite end so that both can be moved up and down to adjust to the proper level for a patient standing upright. This lens is capable of producing a sharp image in an area 3.7 inches in diameter on the 4×5 film. For the best possible results the patient's chest image must be within this area and, therefore, carefully centered.

[1] Presented before the Twenty-fifth Annual Meeting of the Radiological Society of North America, at Atlanta, Dec. 11–15, 1939.

In order to test the capacity of this 4 × 5 film, called the miniature film, it was decided to examine a large number of

Name——————Date——Clinic No.——

0 No findings
0 Suspicious

0 Primary 0 A 0 B
0 Nodes 0 Paren.

0 Pleurisy
0 Dry
0 Wet

0 Minimal
0 Mod. adv.
0 Far adv.
0 Right lung
0 Left lung

0 Excavating
0 Exudative

0 Productive

0 Mixed

0 Apparently healed
0 Calcium
0 Fibrosis

0 Heart
0 Occupational

0 Non-tb. pul. dis.

Fig. 1.

TABLE I.—ACTIVE LESIONS FOUND IN 1,610 CLINIC ATTENDERS EXAMINED ON LARGE AND SMALL FILMS

Diagnosis from Large Film		Disagreement on Small Film	
		No finding	Inactive
Primary active	5	0	0
Pleurisy with effusion	17	0	1
Minimal active	117	5	3
Moderately advanced	92	0	1
Far advanced	40	0	0
	271	5	5

Five were straight misses, but of the five interpreted as inactive one was read "dry pleurisy," two "healed primary," and two "healed minimal." The two "healed primary" would not be re-checked, so they, with the five misses, make seven missed cases, or an error of 2.6 per cent.

patients with both the regular 14 × 17 film and the miniature film, both to be taken at the same visit of the patient to the x-ray department. The regular film represents as nearly as possible the most up-to-date technic. It is taken at 72 inches tube–film distance, using a rotating anode tube, 400 ma., from 60 to 75 kv., and the time on an impulse timer varies from one-fortieth to one-twentieth of a second, depending on the thickness of the patient's chest from actual measurements with calipers. The upper limit of chest thickness which it has been attempted to ray is 30 cm. Patients who had previously had surgical procedures, such as pneumothorax or thoracoplasty, were also eliminated from this study. No other selection was used but most of the patients examined had symptoms of tuberculosis, were exposed to it, or had had a positive skin test. In this study 1,610 cases were so examined, an interpretation of the miniature film being always made and recorded independently and before a similar record of findings was made from the regular film, because the regular film was the standard by which the efficiency of the miniature film must be measured.

For the interpretation of both films a simple card was devised which contained the outstanding points to be recorded. It is here reproduced as Figure 1. The major difficulty with this card is found to be that it requires rather definite classifications and does not provide for the many borderline conditions with which everyone is well acquainted.

A total of 271 patients were found to have active tuberculosis, including the primary type with lymph node enlargement, wet pleurisy, and minimal, moderately, and far advanced cases. In this group, that of primary interest, five of the minimal lesions were missed in the interpretation of the miniature films. Two other minimal lesions were called "healed primary." All of these lesions were very small and might easily have been missed in a regular film taken at a different angle, a slightly different depth of breathing, or with less perfect technic. In nearly every instance these missed lesions were superimposed over rib shadows in the miniature and not obscured in the regular film. Such small lesions may not call for immediate treatment, but should be watched and re-checked for possible changes. Granted that there were five completely missed lesions and two others called "healed primary," making a total of seven missed out of 271 active lesions, there is an error of 2.6 per cent.

The next small lesions to be considered are probably the active primary infection or enlargement of the lymph nodes. Five

such cases were found and none was missed in the miniature film.

The so-called dry pleurisies were made to include all the cases of old pleurisy in which only a small remnant, such as an obliterated costophrenic angle, was the remaining evidence. Of these, nine out of 54 were missed, an error of 17 per cent. This error is unnecessarily large and was greatest in the first group of patients examined because, in the attempt to make a definite recording of the very small lesions and knowing the prevalence of all tuberculous lesions in the upper portions of the lungs, too much concentration was given the apices. In other words, the apices were centered nearer the middle of the film, throwing the bases out of focus. This has since been balanced so that the bases are nearly as well demonstrated as are the apices, with the result that this error can be cut to a much lower percentage with little extra care.

The wet pleurisies included all cases of pleurisy which contained even small amounts of fluid, and none of these was entirely missed. One out of 17 such cases was called "dry" instead of "wet," so the error should not be considered serious.

On the diagnostic card there was no space set aside for "fibrosis of undetermined origin," so it was attempted to de-

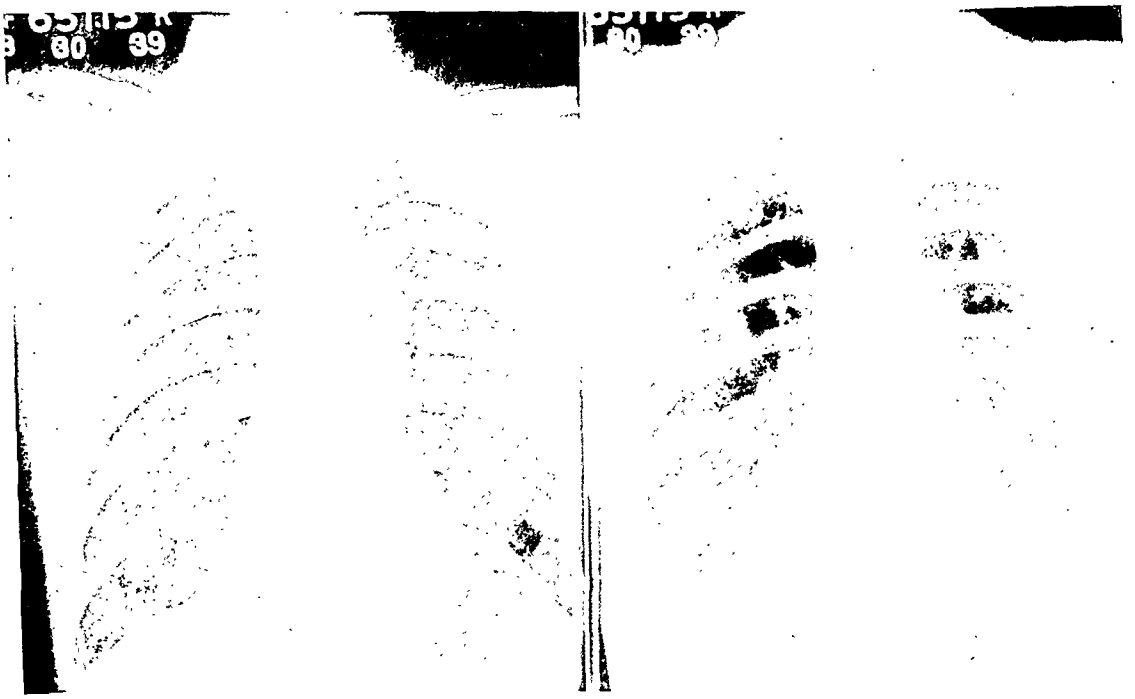
TABLE II.—RELATIONSHIP BETWEEN READINGS OF A REGULAR X-RAY FILM AND A MINIATURE FILM FOR THE SAME INDIVIDUAL

Miniature x-ray film (horizontal columns)	Regular x-ray film (vertical columns) No Find.	Primary A	Pleur. Wet	Minimal Active	Mod. Adv. Active	Far Adv.	Other Lesions	Miniature Totals
No find.	782			5			107	894
Primary A		5						5
Pleur. with eff.			16				2	18
Min. active	3			106	9		2	120
Mod. adv. active				3	78	5	1	87
Far adv. active					4	35	1	40
Other lesions	42		1	3	1		399	446
Large totals	827	5	17	117	92	40	512	1,610

TABLE III.—RELATIONSHIP BETWEEN READINGS OF A REGULAR X-RAY FILM AND A MINIATURE FILM FOR THE SAME INDIVIDUAL

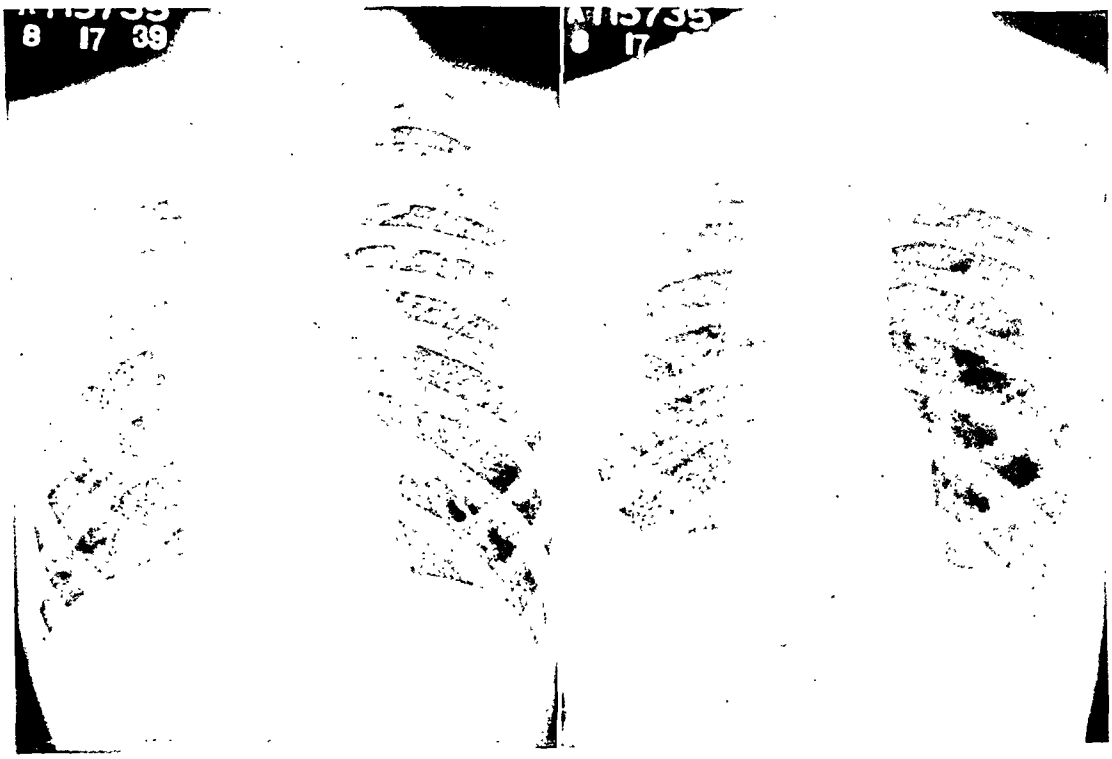
Other than active lesions

Miniature x-ray film (horizontal columns)	Regular x-ray film (vertical columns) No Findings	Suspicious	Primary B	Dry Pleurisy	Minimal Healed	Mod. Adv. Healed	Heart	Non-Tb. pul.	Fibrosis	Pneumothorax	Active	Total Miniature
No findings	782		80	9	7		1	6	4		5	894
Suspicious		1										1
Primary B	31		248		1		1				2	283
Dry pleurisy				43							1	44
Min. healed	2		1		17						2	22
Mod. adv. healed					1	7						8
Heart							10					10
Non-tb. pul.	2							30				32
Fibrosis	7		2			1			30			40
Pneumothorax										6		6
Active lesions	3			2	2	1			1		261	270
Regular film total	827	1	331	54	28	9	12	36	35	6	271	1,610



REG. MIN.

Fig. 2. F-65115, Metastatic malignancy.



REG. MIN.

Fig. 3 A-115735. Far advanced tuberculous lesion with excavation.

cide whether or not that was the result of tuberculosis or some other cause. That is not so easily done, even on the very best large chest film one can produce. From the regular films 28 such cases were classified as "minimal healed tuberculosis." Eight of these were not considered sufficiently significant, in the miniature film, to be mentioned: after a review of errors it is evident that these lesions have no clinical significance. If a descriptive report had been given, it is likely that no significant error would have resulted.

A fair number of the clinic patients were found to have valvular heart lesions from which symptoms often resulted, similar to those found in tuberculosis. The cardiac shadow in the small film is good because it is in the center of the film, where the greatest sharpness of detail is produced, but it was noticed that the heart shadow appeared enlarged in the small film. Therefore, rather than measure the diameters, particular account of the shape was taken. Two in 12 such cases were missed but, upon reviewing the errors, they were found to be entirely oversights because the findings were definitely there.

As regards non-tuberculous lesions and especially those at the base, the same things can be said about these conditions as were said about dry pleurisies. Some were missed at first, while too much concentration was given to the apical areas. Basal detail can just as well be obtained and its interpretation secured when one has learned to distinguish between shadow differences in the miniature and in the regular films.

In a critical analysis of any size of small film it must be remembered that both normal and abnormal conditions within the lungs are necessarily crowded into a smaller space and detail must of necessity suffer, whether one enlarges the film or views it as it is. It also naturally follows that the smaller the film the greater is this loss. An attempt has been made to demonstrate this point by making lantern slide reductions of both the regular film and the miniature film made of the same patient at the same visit to the department. However good the small film may be, it lacks the richness of detail of the regular film made with up-to-date equipment, as mentioned before.

Having established the reasonable accuracy of the method, it is now being



Fig. 4. I-44473, Minimal tuberculous lesion.

applied to every new patient coming to the tuberculosis clinic, without the taking of a large film save in cases in which there is some questionable appearance in the small film. This has proven satisfactory in the two months this procedure has been followed, and obviously it is a great saving in film cost. There have been 1,719 persons examined in this group.

As a survey of presumably well persons who might have tuberculosis among their number, two groups are now being examined with the miniature film. The first is made up of the women attending the prenatal clinic of the health department. These are women unable to afford a private physician and, because of their age group and the fact that they are pregnant, it is important to examine them for tuberculosis—for their own safety as well as that of the expected child.

The second group of well persons being surveyed is made up of women who are employed on a WPA project in which the employees go out as housekeepers in welfare families where additional help in housekeeping is needed. It is important to examine these women to be sure that they are free of tuberculosis before they go to work in families with children.

A tuberculin test is being done on many of these women, using a single dose of 1 to 1,000 dilution intracutaneously, and all are x-rayed regardless of the result of the test. This will, in time, give valuable data on the value and accuracy of the prescreening with tuberculin. This will be reported in a later paper, since only 754 women have been examined so far, of whom four have been found with significant lesions.

In proposing this method of examination it seems that it can be carried out economically enough to make it possible to apply it directly in surveying large numbers without a preliminary tuberculin test, even in groups in which the reactor rate is low. In addition, there are certain inaccuracies in tuberculin testing, especially when only one dose is used, a 1 to 1,000 intracutaneous dose, or the von Pirquet, which approximates this dose in sensitiveness. In a recent study by Musacchio (8), an associate, in which he tested 1,000 cases of tuberculosis, it was found that, using a 1 to 1,000 dilution intracutaneously, there

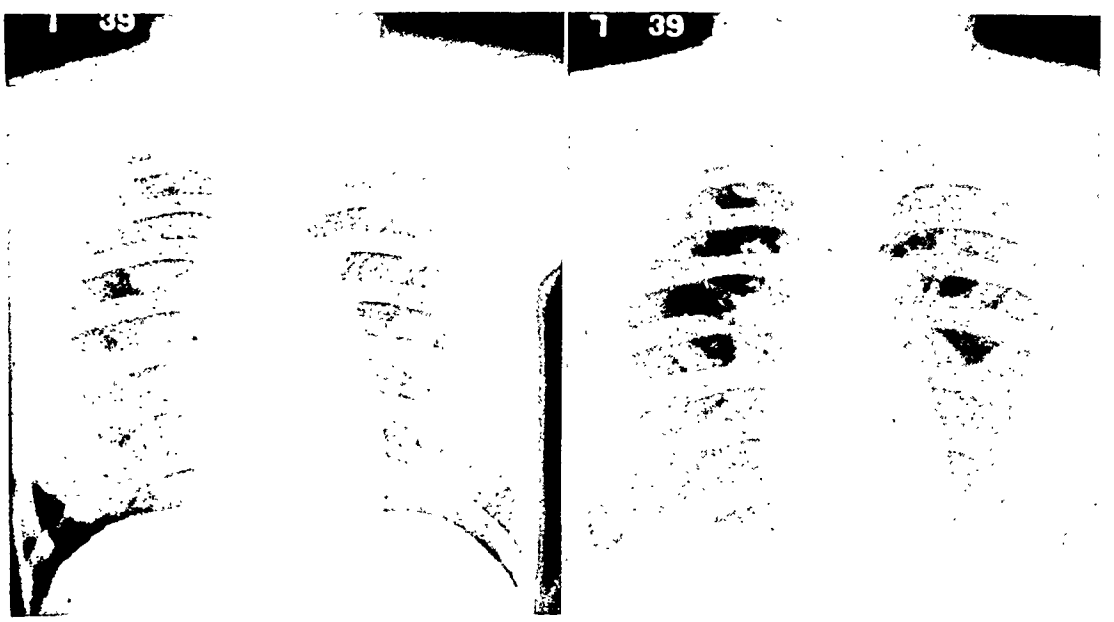


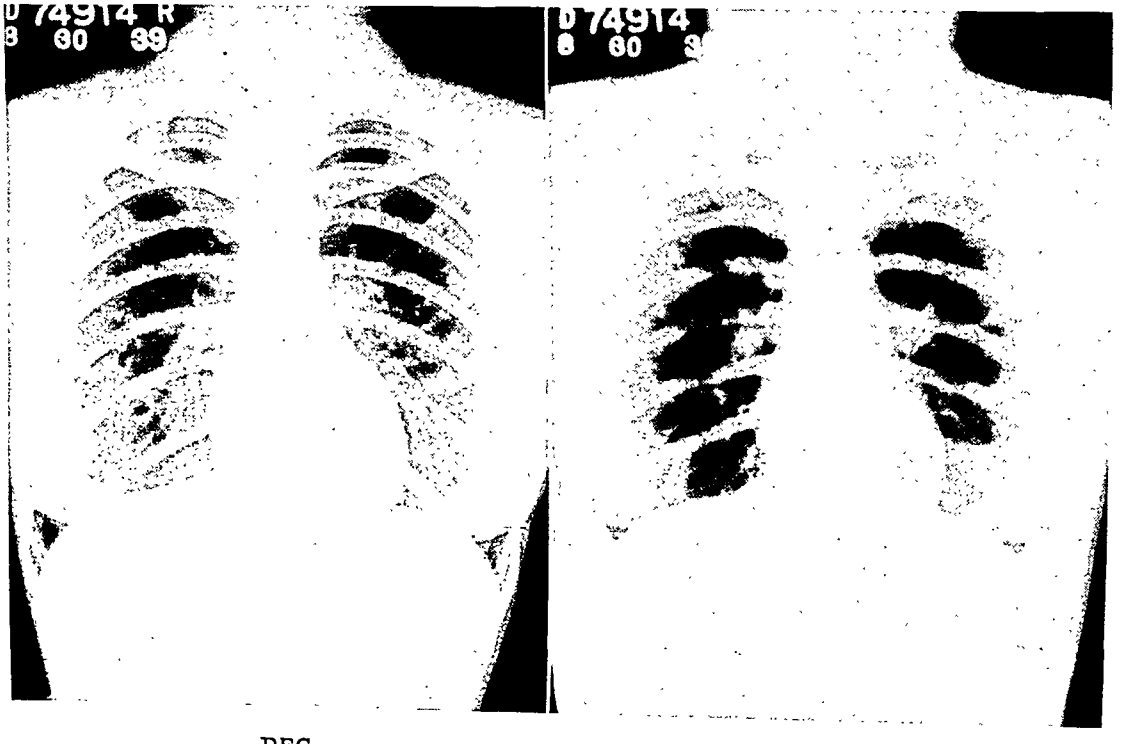
Fig. 5. H-49931, Minimal tuberculous lesion.

were 847 reactors (or 84.7 per cent) and then, retesting with 1 to 100 dilution, there were added 122 (or 12.2 per cent) more. This is a significant number to miss with a one-dose method, and, of course, if a second test is given, it means extra effort and expense.

In using the x-ray alone there is need for only one visit by the patient, at which time the examination is made which results in a permanent record. In most instances this is complete, only a few patients needing re-examination; whereas with the preliminary tuberculin test, one visit is required to make the test, another for the reading, and if the two-dose method is used, still another visit is required before the x-ray is made. There is always a loss growing out of the fact that many reactors fail to appear for x-ray examination. This amounted to 4,719 out of 31,803 reactors (or 14.8 per cent), in a recent study in Detroit (9). Add to this the fact that most persons would rather have a roentgenogram taken than submit to a tuberculin test. There is also the possibility of the detection of other nontuberculous lesions of the heart and lungs which would be missed if only a tuberculin test were made. It is apparent, then, that there are several good reasons why a reasonably accurate but economical x-ray examination is better applied directly than when tuberculin is used as a preliminary screen.

The cost of taking the 4 × 5 inch film is very reasonable. In a test run 73 persons were x-rayed in 65 minutes, and the development of the films can be carried out as rapidly as they are exposed. To register, supervise the dressing room, place the patient, make exposure, and develop the film required the time of six persons.

The cost of a 4 × 5 film is approximately one-tenth of the cost of a 14 × 17 film, so it represents a very material saving. The tube cost for an exposure of a miniature film is nearly the same as the cost of exposure of a regular film when a rotating anode tube is used.



REG. MIN.

Fig. 6. D-74914, Minimal tuberculous lesion—right.

These small films are easily handled, can be interpreted at the same speed as the large films, and require little space for storage.

To compare this method with other x-ray examination methods that may be used for direct survey purposes, there are four which should be considered.

First, full size celluloid film taken with a good technic is accurate but the cost makes it prohibitive for extensive use in large surveys.

Second, there is sensitized paper which, while reasonably accurate and about one-half as expensive in material cost as full size film, is still too expensive and is bulky to handle and store.

Again, the fluoroscope has been used extensively for surveys and, while quite inexpensive, its accuracy is open to some question, since no permanent record is made except in those cases referred for film examination.

Fellows (10) and his associates who have used the fluoroscope extensively and have made careful comparisons with well taken full size films found an error of 13 per cent in missed pulmonary tuberculous lesions in a series of 2,300 persons both filmed and fluoroscoped, taking the film as 100 per cent. The miniature film, 4 × 5 inches in size, can do definitely better than this.

Finally, the 35 mm. film, sometimes called "micro film," the actual field being 25 mm., while quite inexpensive to produce, is, in the opinion of many, too small. It must be enlarged either by projection or printing in order to permit of interpretation and in this enlargement considerable detail is lost. While this method has not yet been subjected to careful comparison for accuracy with full size film and with 4 × 5 inch film, some observation of its use suggests that too much has been sacrificed in accuracy in favor of economy, which cannot be said of the 4 × 5 inch film.

SUMMARY AND CONCLUSIONS

As a result of this study the authors believe they are justified in drawing the following conclusions regarding photoroentgenography with the 4 × 5 inch film:

1. This experience indicates that the

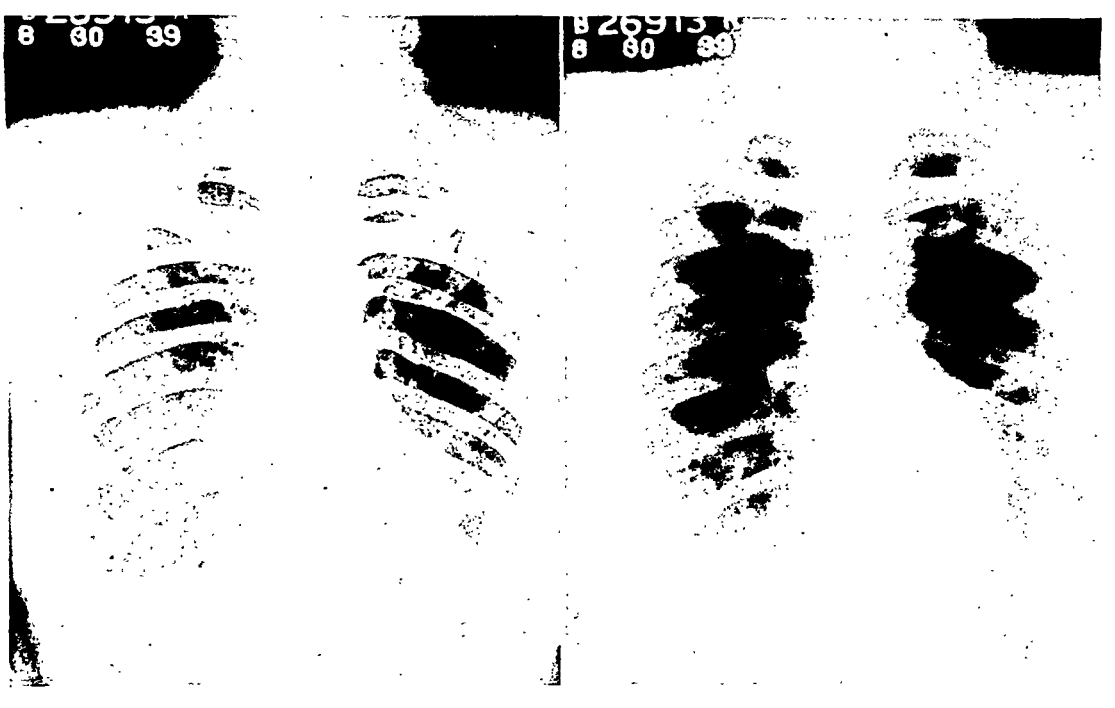


Fig. 7. B-26913, Minimal tuberculous lesion—left.

4 × 5 inch photoroentgenograph is quite accurate. (Only 2.6 per cent error in detecting 271 cases with active tuberculous lesions as found in the full size film taken of 1,610 persons.)

2. This type of examination can be applied in survey work in tuberculosis to large numbers rapidly and economically. (Slightly faster than one per minute at a material reduction in cost.)
3. It has definite advantages over certain other x-ray methods of examination either in accuracy or in cost or both.
4. That this method of examination can be applied directly without preliminary tuberculin testing with greater accuracy and at the same time economically.
5. Finally, the authors believe photoroentgenography of this type offers a practical method for case finding in tuberculosis for use in surveying large numbers in susceptible groups.

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THE ROENTGENOGRAPHIC PROGNOSIS OF PULMONARY TUBERCULOSIS

By J. GERSHON-COHEN, M.D., *and* JOHN T. FARRELL, JR., M.D., *Philadelphia*

DESPITE the declining incidence of tuberculosis, the mortality rate continues to remain about the same. It has been estimated that approximately 50 per cent of the patients who develop clinical disease succumb, in spite of the aid of modern therapy. It is well, then, for the roentgenologist to investigate the prognostic possibilities of the roentgen examination of the chest which has now become a procedure of prime importance in the diagnosis of pulmonary tuberculosis. Can he foretell into which of the two groups, almost equally divided, any one case will fall? Immediately it becomes apparent that some standardization in diagnosis will have to be developed before any system of prognosis can be set up. To this end, the system of diagnosis adhered to, was arranged as follows:

THE ROENTGEN DIAGNOSIS

The essentials noted are:

1. Extent and distribution of lesions.
2. Type of lesions.
3. Cavities.

1. The extent and distribution of lesions are divided roughly into minimal, moderate, and widespread. Minimal, if less than two interspaces; moderate, about one lobe; widespread, if more than one whole lobe is involved.

2. The lesions are typed either predominantly as calcific, fibroid, exudative, caseous, or cavernous. When there are combinations of these types, the whole combination is listed in the order of their prominence, *viz.*, fibro-caseous, fibro-calcific, cavernous-fibroid, or caseous-cavernous, etc. The character of infiltration precedes the designation of the type, being described as diffuse, dense, linear, or discrete; if discrete—whether miliary, submiliary, acinar, acino-nodose, nodular, tuberculomatous, patchy bronchopneumonic, or lobar. This will thus result in such designations as a "minimal linear fibroid" infiltration, or a "moderate disseminated bronchopneumonic exudative," or a "widespread submiliary fibro-calcific infiltration."

3. Cavities receive special diagnostic consideration. The diameter is measured. The character of the walls and of the perifocal reaction is studied to learn whether exudation, caseation, or fibrosis predominates. Other important diagnostic criteria to be appraised are the completeness of drainage from the cavity, the extent of symphysis of the cavity wall with the parietal pleura, and the location of the cavity with relation to the lobes. These facts are often ascertained only from many films made with different angles of projection. It is sometimes even necessary to wait until after pneumothorax before arriving at a final conclusion.

ROENTGEN PROGNOSIS

After this methodical recording of the roentgen diagnosis, a prognosis is made, according to the following system. The forecast is limited to each lung separately; the changes on one side, however, bear on those of the other side in a given relationship worked in as part of the system.

BENIGN GROUP

Benign 4. The lesions are *most benign*, healed, or shall heal within four months.

Benign 3. Not quite *most benign*, and

[1] Presented before the Twenty-fifth Annual Meeting of the Radiological Society of North America, at Atlanta, Dec. 11–15, 1939.

the lesions shall heal within eight months.

Benign 2. Not quite *least benign*, and the lesions shall heal within 12 months.

Benign 1. The lesions are *least benign* and take as long as 18 months for complete resolution and organization.

VIRULENT GROUP

Virulent 1. These lesions are *least malignant* and heal within 18 months.

Virulent 2. Not quite *least malignant* and healing occurs within 24 months.

Virulent 3. Next to *most malignant* and healing may take 36 months.

Virulent 4. These lesions are *most malignant* and are hopeless, except in those cases in which surgical intervention may be successful. The remainder either terminate as so-called "good chronics" or die within a few weeks or months with terminal manifestations of the disease.

Benign 4.—These lesions are minimal in extent, fibroid or calcific. The distribution may be linear or discrete. In some cases, only some thickening of the pleura or pleuritic adhesions may be present in the later stages, or a simple idiopathic pleural effusion may be the only manifestation. These patients present few clinical symptoms, most of which are only suggestive; such as fatigue, failure to gain weight, or other such clinical symptoms of tuberculosis. There is no cough or expectoration. If sputum is present, no tubercle bacilli are found. In many cases, one sees only a few residua of hematogenously spread tuberculosis in which complete screening has taken place in the pulmonary capillary bed. This form may often be regarded as part and parcel of the primary complex, even though the earmarks are those of re-infection. It is usually impossible to be sure when the changes occurred with relation to the primary infection.

Benign 3.—This resembles the Benign 4 classification, but the lesions are more extensively disseminated, either in a localized portion of a lung or scattered throughout both lungs, but never involving an area greater than that covered by two interspaces. These lesions, too, are often the residue of a hematogenous spread of the limited, protracted, and disseminated types. Fibrosis and calcification predominate. There are practically no chest symptoms and if cough is present, it is usually nonproductive. A few suggestive symptoms, such as fatigue or underweight, may be present.

Both Benign 4 and Benign 3 are to be considered pre-clinical phases of adult tuberculosis and are often regarded as manifestations of the primary infection or of the second stage of hematogenous spread, organization of which is moderately advanced.

Benign 2.—Here, the lesions are minimal in extent, predominantly fibroid, either linear, fine-grained, or discrete in type. While the sputum is negative for the presence of tubercle bacilli, some definite clinical symptoms or signs of tuberculosis have developed, usually hemoptysis, fever, râles, and a slight increase in the blood sedimentation rate. This state is to be regarded as the earliest and most benign form of clinical adult phthisis in which recovery, by rest alone, is the rule within 12 months.

Benign 1.—This is like the preceding stage except that the lesions are more extensive, but do not exceed a moderate degree. Not more than the total area of one lobe is involved in the postero-anterior film. Fibrosis, though not necessarily organized, predominates. The linear fibrosis is not so fine-grained as in Benign 2. The discrete lesions are also fibrous, but not organized. *No cavities* are present in this group and the sputum must be scant, containing few, if any, tubercle bacilli. The clinical symptoms may be definite, but not marked. While clinical adult

tuberculosis is well established in this stage, very little bronchogenic spread occurs; no caseation or cavitation develops and, with absolute rest, the lesions show a tendency to heal usually within one year, but at the outside in 18 months.

The benign forms are thus split into two sections: the pre-clinical and clinical forms of tuberculosis, but all four groups are essentially of the fibroid type.

Virulent 1.—In this stage, the lesions are still minimal in extent but, though predominantly fibrous, evidence of proliferation, exudation, or caseation is already present. There are no cavities present. Bronchogenic spread may have occurred. Bacilli may occur in the sputum, but not in excessive amounts. The clinical symptoms are of recent origin and the patient has a good chance of recovery, with conservative treatment, within 12 months, although organization of the lesions may take 18 months.

Virulent 2.—This resembles the above stage, only the lesions may be moderate in extent and a cavity less than 2 cm. in diameter may be present. While some fibrosis may exist, exudation or caseation predominates. Into this group are also placed those cases of widespread involvement, if most of the infiltration is fibrous. The symptoms are of recent origin, and not more than six months in duration. Possibly 50 per cent of these patients will get well on conservative measures and this presumes disappearance of the cavities. A good portion of the other 50 per cent get well by pneumothorax or surgical intervention, if begun without much delay. These cases have full-blown symptoms and signs of fibro-cavernous tuberculosis or caseous-cavernous tuberculosis. Healing is expected to take place within 24 months.

Virulent 3.—These cases actually fall into the far advanced group, and could be regarded frequently as terminal were it not for the advantages of pneumothorax and the newer surgical procedures now available. Some few of these cases heal with conservative therapy and a small percentage become "good chronics." In all cases of this group, the phthisis is well established and well organized, the usual appearance is that of a diffuse exudative cavitary phthisis. The cavity is not over 5 cm. in diameter and if there are multiple cavities, their combined volumes should not exceed that of a cavity 5 cm. in diameter. Areas of exudation or caseation are irregularly distributed in the parenchyma, but evidence of exudation or caseation is always present in the perifocal areas about the cavities. If no cavities are present, but if there is widespread involvement of the lungs, even if an exudation or caseous response is present in only a few lesions or places, then such cases are also classed in this group. The sputum is usually, but not always, heavily positive. The pleura is often thick and involved with many adhesions so that when pneumothorax can be done, it often interferes seriously with compression of the cavities. Other surgical measures must be resorted to, and it is only because of their effectiveness that these cases are put in this category instead of being placed in the next or most virulent group. To prevent confusion, it should be restated that in the presence of excavation, the extent of parenchymatous involvement must not exceed a moderate degree, the only exception being those cases in which *no cavities* are present when the involvement of the parenchyma may be widespread and extensive.

Virulent 4.—All the so-called hopeless cases fall into this group, in one of three ways:

(*a*) A large chronic fibrous-walled cavity (over 5 cm. in diameter) may be present which can be dealt with only by thoracoplasty and, even then, not always successfully.

(*b*) The cavernous lesions may be so old and well established that even when they do not ulcerate with progressive enlargement, at best they remain almost stationary, the case coming under that heading designated as a "good chronic."

(*c*) The lesions may be so widespread, exudative, or caseous in nature as to leave

no doubt of the terminal nature and outcome of the disease.

Re-classification of Bilateral Lesions.—The above classification holds when only one lung is involved and this must be constantly kept in mind. When bilateral lesions are present, automatic shifts take place in this classification; by this is meant that the prognosis is worse and is so indicated according to a prescribed method of re-classification.

BILATERAL BENIGN LESIONS

1. If the original prognosis falls into the benign group on both sides, a shift to the left is made for each side. For instance, if the prognosis is Benign 3 right and Benign 2 left, then the final classification becomes Benign 2 right and Benign 1 left.

VIRULENT ON ONE AND BENIGN LESION ON THE OTHER SIDE

2. If the lesions are virulent on one side and benign on the other, then the benign designation, no matter of what grade, automatically becomes Virulent 1, but there is no change made in the original virulent designation of the other side. For instance, if the prognosis is Virulent 2 on the right and Benign 3 on the left, then the new classification becomes Virulent 2 on the right (unchanged) and Virulent 1 on the left.

BILATERAL VIRULENT LESIONS

3. If the lesions are in the virulent category on both sides, the shift to the left is done by adding the grade of the contralateral side to the original grade. For instance, if the classification on the right is Virulent 2 and on the left Virulent 3, then the final grading would become Virulent 5, but since these totals exceed 4, we simply call each side Virulent 4. If the total of both sides is less than 4, the simple totals hold. If the totals are exactly 4, we allow this to stand if this hopeless prognosis seems warranted by the clinical picture, but if the clinical picture is more favorable, we allow these cases to fall back into Virulent 3 for each side.

The technical exceptions for re-classification are very important and must be followed rigidly if good results are to be expected from this system.

COMMENT

Over 600 cases have been analyzed at the Eagleville Sanatorium, Eagleville Dispensary, and White Haven Sanatorium and the preliminary estimate reveals that our prognoses were correct for approximately 80 per cent. And, in order to limit the analysis as much as possible to the roentgen examination, the clinical picture and laboratory findings were excluded from all prognostic considerations. We believe, therefore, that when the same cases are reviewed to include these additional data, a better average of accuracy will be achieved.

Our best prognoses were made in the more advanced benign and virulent groups. In the Benign 1 and Virulent 1 groups, our failures were more pronounced, but it so happens that in our material, the smallest number of cases fell into these groups. Our good general average of accurate prognoses was thus little influenced. In the sanatoria, most of the cases were benign or virulent to a moderate or advanced degree, but, to our surprise, the same held true essentially in the dispensary practice. This would seem to indicate again that by the time the roentgen examination is asked for, pulmonary tuberculosis has passed beyond the early stages in which event none of us, the roentgenologist included, can foretell from a single examination which way the disease will progress. Resort to serial examinations can be made, however, and a fairly accurate prognosis arrived at in a few weeks. The statistical analysis of this work is to form the basis of a separate report to be given at an early date.

Dr. Chamberlain knows all about the third exception, coccidiomycosis. Fortunately we do not have that in New England: I do not believe Dr. Doub had any in his group of cases. It is interesting to note the great number of cases which have been reported recently as "valley disease" which are, as far as I can learn, cases of pulmonary coccidiomycosis which have an acute onset, a short course, and complete recovery. A similar infection with a similar organism when there are also bone, skin, or gland lesions is usually fatal. Why you get that wide variation I do not know.

Finally, the monilia which has been emphasized repeatedly as the cause of pulmonary disease has, in our experience, invariably been the secondary invader. We are unable to find a single case which had monilia in the sputum that had not an underlying lesion such as carcinoma of the lung, bronchiectasis, or tuberculosis which could perfectly well explain the presence of a secondary saprophytic invader.

Aspergillus, I believe, causes little in the way of symptoms. Two of the United States Public Health men in Pitcher, Oklahoma, reported 120 cases of what was presumably a healed aspergillus infection in the lungs of miners, and they could not obtain a history of any illness in the majority of these men. The "pigeon breeder" who gets this disease should read, I think, "pigeon feeder," because in the Rhineland of Germany men have a very unhappy type of occupation in stuffing squabs for market. They take young pigeons and to get them fat rapidly, these men chew up grain, masticating it to a soft consistency, and then force it into the squab's beak with their lips, so the "pigeon feeders" get this disease rather than the "pigeon breeders." I do not believe any of our people here in the States will get pigeon feeders' disease.

Finally, I would like to interpolate a word about discussions. Too often there is not enough time available for discussion. Honest criticisms and frank differences of opinion often crystallize one's ideas and may add considerably to the value of a presentation such as this series of papers. I apologize if I have hurt anyone's feelings; there is nothing personal in my random remarks. The *argumentum ad hominem* must always be avoided in these meetings. May I compliment Dr. Doub on the symposium which he has arranged.

COL. ALBERT BOWEN (Fort McPherson, Ga.): We have been much interested in the use of the term "pneumonitis" to designate an acute, contagious form of respiratory disease manifested by fever, leukopenia, malaise, etc., usually quite mild, with definite, circumscribed x-ray evidence of exudation and possibly infiltration.

Since we first reported epidemics of acute influenza pneumonitis, in Hawaii, in 1934, there has been an increasing number of reports of epidemics of "benign," "circumscribed," "leukopenic," "atypical," "virus," and "interstitial" pneumonitis. Each reporter has chosen his own modifying term and they all seem to apply to this disease.

Those who have seen these epidemics are convinced that the disease is a clinical entity. Those who have seen only films of the cases say it is just a respiratory infection or bronchopneumonia. I have received personal communications reporting epidemics which have not appeared in the literature. Baylor University Hospital had an epidemic of some sixty cases, over a year ago, which has recently been reported in the *Texas State Jour. Med.* Wingfield, at Antioch College, has observed pneumonitis cases for several years and makes the interesting report that there is no change in the incidence of this acute benign pneumonitis during the influenza epidemics which occur from time to time.

The dispensary staff, at Cornell University, reported in the *Jour. Am. Med. Assn.*, last May, their experience with epidemics of what they termed "acute interstitial pneumonitis," thus being at variance with Dr. Hodges' definition of

interstitial pneumonitis as being only chronic.

We have been inclined to agree with Reimann and some of the other observers that this is probably due to a virus, inasmuch as the typical cases all show leukopenia and show no pneumococci or other constant bacterial findings.

It seems to us that we are dealing with a definite clinical entity and the problem of what to call it, I think, can well await the discovery of its etiology.

JOHN T. FARRELL, JR., M.D. (*closing*): The most important thing in the practice of medicine, as far as the patient is concerned, is the diagnosis, but in many conditions, as far as the family is concerned, the most important thing is: what is going to happen to the patient? In a disease like tuberculosis, the economic aspect, the question of insurance, the question of continuing work, the question of marriage, all enter in and we feel that anything which helps to give a more accurate prognosis is of value.

As to the terminology, I would agree that the term "malignant" should be reserved to neoplasm, in spite of the fact that we use the term "malignant hypertension." Possibly it would be better to simplify the terminology and speak of a favorable I, II, III, IV, or an unfavorable I, II, III, IV.

From our own experience we know that bilateral lesions behave differently from unilateral. It might be possible, in a scheme of this kind, to reduce it to the terms of contract bridge bidding, assigning a number for involvement of one lobe; a larger number for the involvement of two lobes, presence of cavitation, caseation, and other numbers depending on the extent and character of the disease so that one would have a numerical basis for the determination of the prognosis.

We have recognized the fact that in a particular instance a single film is often not enough to determine what the prognosis is and we have advocated the use of serial studies.

We realize that there are many other factors—age, race, and the presence of diabetes, which have a good deal to do with prognosis, but we were trying to devise a method which could be applied by the roentgenologist. The value of this report lies in the fact that the prognosis is practically limited to the x-ray findings. If to these one adds the implications of other factors such as age, race, and complications, a prognostic accuracy of more than 80 per cent, which was achieved by the roentgen indications alone, should be possible.

HOWARD P. DOUB, M.D. (*closing*): I wish to say that Dr. Sosman and I are not much at variance in our statements about bronchomycosis. I believe that, in most instances, these mycoses are saprophytes but there are certain cases in which they are primary invaders. In several of our cases, in which we had autopsy confirmation, I believe the mycotic infection was the cause of death.

In coccidioidal granuloma, which occurs mostly in California, it is very definitely a primary infection.

EVALUATION OF ROENTGEN THERAPY IN SINUS DISEASE[1]

By J. R. MAXFIELD, JR.,[2] A.B., M.D., Instructor in Radiology, *and* CHARLES L. MARTIN, E.E., M.D., Professor of Radiology, Baylor Medical College, *Dallas, Texas*

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ALTHOUGH the medical men in ancient times did not have the same conception and understanding of the nasal sinuses that we have to-day, they did make references to conditions which, in the light of our present knowledge, can be interpreted as sinus disease. Since Albright (1) has thoroughly covered the history of sinus disease from antiquity to the present time, it is sufficient to say here that until 1850 our modern knowledge and conception of sinus disease were unborn.

From the period of 1850 until 1900, rapid steps were made in the understanding of the anatomy of the bones of the face and of the paranasal sinuses. In this period the names of Caldwell and Luc (23) came into prominence. Caldwell advised making an opening through the canine fossa and a counter-opening through the inferior meatus. The primary opening was allowed to close and treatment was carried on through the opening in the meatus. In 1903, Luc described a similar approach and advised complete removal of the lining membranes. The accomplishments of these men marked the beginning of radical nasal surgery which reached its height about 1915.

Surgeons and rhinologists of this era from 1850 until 1915 were attempting to relieve the pathology in the accessory sinuses by surgical procedures, that is, by attempting to modify the anatomy of the accessory nasal sinuses. Their byword was drainage, drainage, and more drainage. When their surgical procedures failed, more radical ones were advised in an attempt to turn failure into success by more surgery. The end-results in most instances, however, were most unfavorable. The adage grew, and was true, that sinus surgery—once performed—required sinus surgery from then on.

About 1913 there began a metamorphosis from the radical opinion held by the rhinologists to one of conservatism. Pathology in the accessory sinuses was being studied in an attempt to explain their diseases. The consistent failure of the radical procedures forced the surgeons to turn to physiology and biology for an explanation of "why the surgery failed." The consensus of opinion of the more conservative, better trained men in otolaryngology is expressed by the following quotation from Wright (23):

> "The period of my activity, covering as it does more than 25 years, witnessed the absurd exaggeration of notions as to the frequency when sinus disease required surgical intervention and the rash resort to this devastating destruction of nasal structures in operative measures designed to afford relief to affections more or less trifling in themselves. It experienced the disappointment of too enthusiastic operators and too credulous patients. It saw reputations, world-wide in the domain of our specialty, made out of a manual dexterity perhaps, but also an intrepidity of spirit which often approached too much upon the rights of humanity and too often disregarded the precepts of conscientious professional contacts."

By 1915 a better understanding of the function of nasal sinuses in health and disease was being manifest. The action of the cilia in the sinuses was demonstrated to be a protective mechanism. The names of Bloomfield, Yates, Hartz, Hilding, Pretz, and others appeared in the literature. Attempts were made to impress the rhinologists with the importance of the protective mechanism present in all si-

[1] Read before the Texas Radiological Society at the Annual Meeting in San Antonio, Oct. 22, 1938.

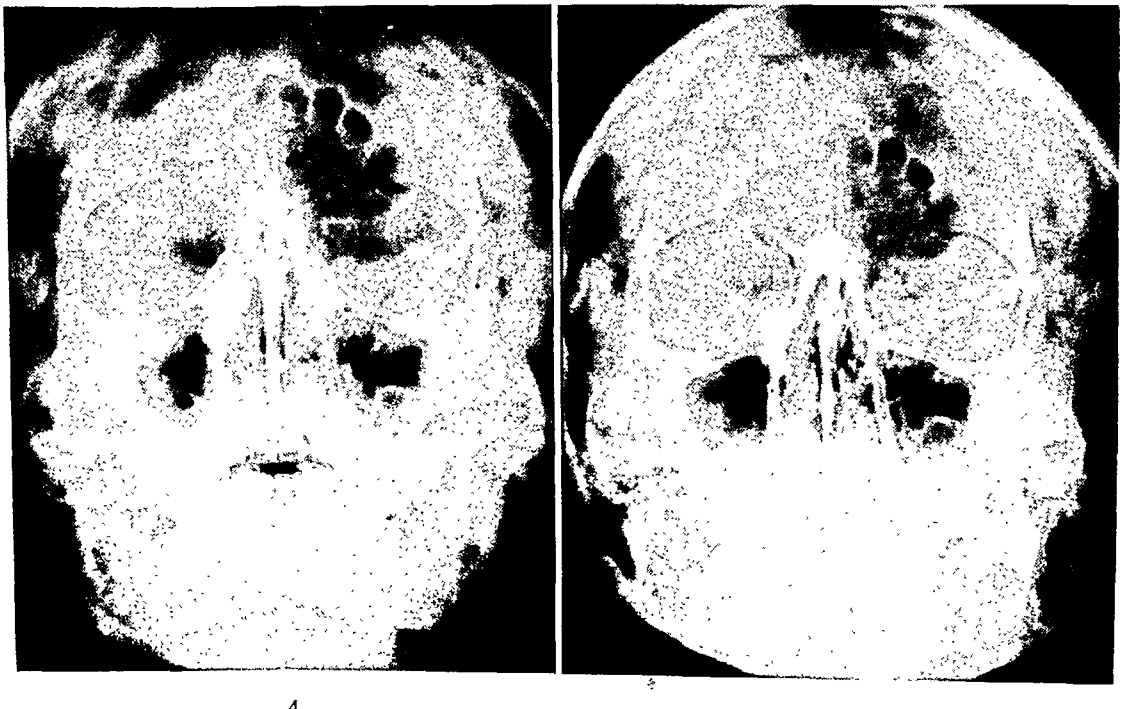
[2] Now Instructor in Roentgenology, University of California Medical School, San Francisco.

nuses. Operators began to realize the importance of the preservation of the normal protective mechanism instead of attempting to correct the defects of Nature in anatomical structures, and thereby hoping to secure drainage by gravity, but the day of radical surgery had not passed. New methods of attack from an anatomical standpoint were reported and countless hundreds of patients were being subjected to radical sinus operations month after month and year after year. This condition is still present in our modern era but, fortunately, a more marked trend toward conservatism is manifest than at any time in previous years.

Even as late as the past decade conflicting reports can be found coming from the same institutions as to the values and results of surgery on the accessory nasal sinuses. Hilding (8), at the Mayo Clinic, reports that the results of operation on the accessory nasal sinuses in general are not entirely satisfactory. With this in mind his series of experiments on the physiology of the nose and sinuses were carried out in the hope of finding possible causes for failure of operations on the sinuses. From this same institution and covering the same general period is the report of Williams (21), in which he says that 200 cases, studied at the Mayo Clinic, operated on in 1926, showed 84.6 per cent cases of symptomatic relief and

TABLE I.—RESULTS REPORTED FROM SURGERY

	Cured	Improved	Unchanged
Barlow	47%	30%	23%
Tucker	99%	1%	0
Stevenson	94%	3%	3%
Williams	84.6%	12.4%	3%
Thorburn and Patozzi	43%	35%	22%
Richards	34%	20%	46%



A *B*

Fig. 1. Miss H. H., white, nurse by occupation, aged 26 years. Symptoms of three months' duration—purulent discharge, severe headaches, morning cough—no relief from conservative treatment.

Fig. 1-*A*. Roentgenograph made Dec. 1, 1937, reveals proliferative soft tissue in lateral portion of right antrum, with increased density in right frontal sinus. X-ray therapy: Dec. 3, 1937, 400 r to sinuses.

Fig. 1-*B*. Roentgenograph made *Jan. 3, 1938*, shows sinuses clear. Complete symptomatic relief. Remained well for 11 months, then, following an acute cold, developed symptomatology and radiographic findings similar to those found on first visit. Radiation therapy (Nov. 19, 1938) gave relief and the patient has continued well until the present time.

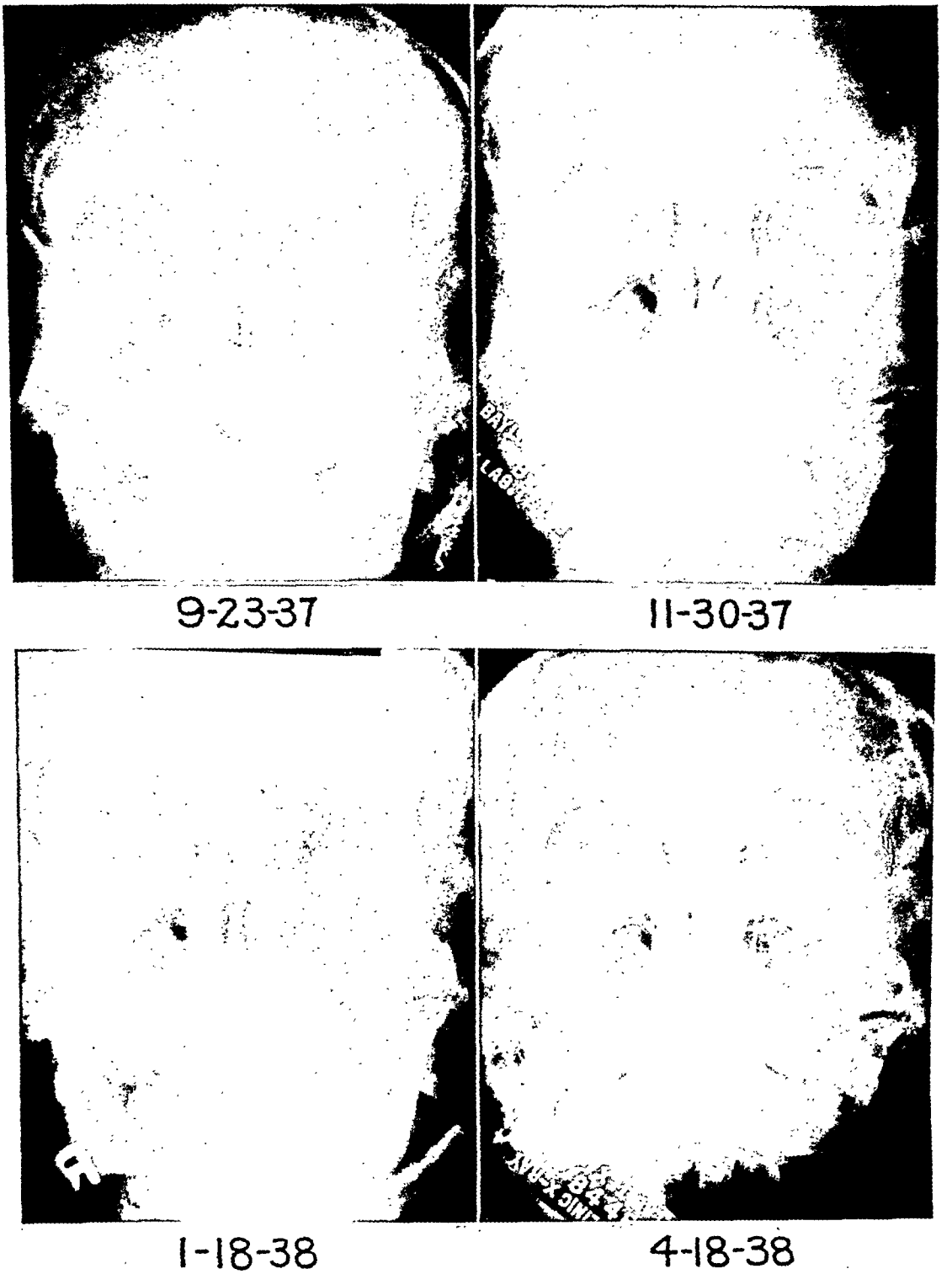


Fig. 2. D. H., white, male, aged 13 years. History of sinus disease for several years. Intensive treatment by allergy department, nasal packs, nose drops, etc., for six months without relief.

Fig. 2-*A*. Roentgenograph made Sept. 23, 1937, shows proliferative soft tissue in both antra. Roentgen therapy (300 r).

Fig. 2-*B*. Roentgenograph made Nov. 30, 1937, shows definite improvement: all symptoms relieved. Jan. 2, 1938, for check-up routine examination; no complaints; doctor prescribed nose drops for dry nasal

only 6 per cent that required a second operation, "the condition having cleared up not only from the patient's standpoint but from our standpoint as well. These results should tend to change the opinion which is sometimes expressed by the uninformed that it is difficult to secure symptomatic relief in chronic sinus disease, at least insofar as the maxillary sinus is concerned." With two such reports from the same institution, it is little wonder that the general practitioner or the oto-laryngologist in the smaller centers is in a quandary as to which to follow. The results shown in Table I also tend to emphasize the great variation in the results of treatments in various institutions. It does bring one to feel very definitely that the answer to the sinus problem is not yet at hand and that radical surgery is not the answer to the handling of all sinus disease. These few brief quotations will sum up the period to the present time.

Jervey (9), in 1935, said: "Suffice it to say that to-day the trend of radical surgery is on the wane, though we still hear of interns at special hospitals who boast of the number of 'radicals' which they have done without, unfortunately, a check-up on end-results." He stresses that an attenuated sinus cannot be restored to a normally functioning sinus.

Hays (7), in 1937, said: "With the attendance upon thousands of patients with nasal complaints in over 25 years of specialized practice, I believe I am in a position to state that the majority of prospective sinus conditions should not be operated on and that almost without exception the majority of sinus infections will recover if treated conservatively."

Snow (17), in 1937, said: "Sufferers from sinusitis want relief from pain and discomfort . . . granted that surgical intervention is occasionally necessary, operative procedures fall far short of being satisfactory as attested to by patients and internists alike."

Buckley (3) said: "When operation is done, regardless of the kind, the nose is mutilated to a certain degree and unless the result is going to more than compensate for the mutilation, we are not going to do any good but we are actually hurting our patient."

Salinger (14), in 1937, said: "The literature in 1936 reveals nothing revolutionary or startling. It is nevertheless significant in demonstrating a saner appreciation of basic anatomic and physiological data and manifesting a more conservative medical and surgical treatment."

The surgery of the accessory nasal sinuses has not been entirely satisfactory; so likewise have failures accompanied other methods of treatment that have been introduced. The main point in favor of some of the conservative methods of treatment over surgery is that they do not damage the patient as much. It is universally accepted that poor medical results can quite easily be supplemented by surgical procedures, but poor surgical results cannot easily be corrected by medical treatment.

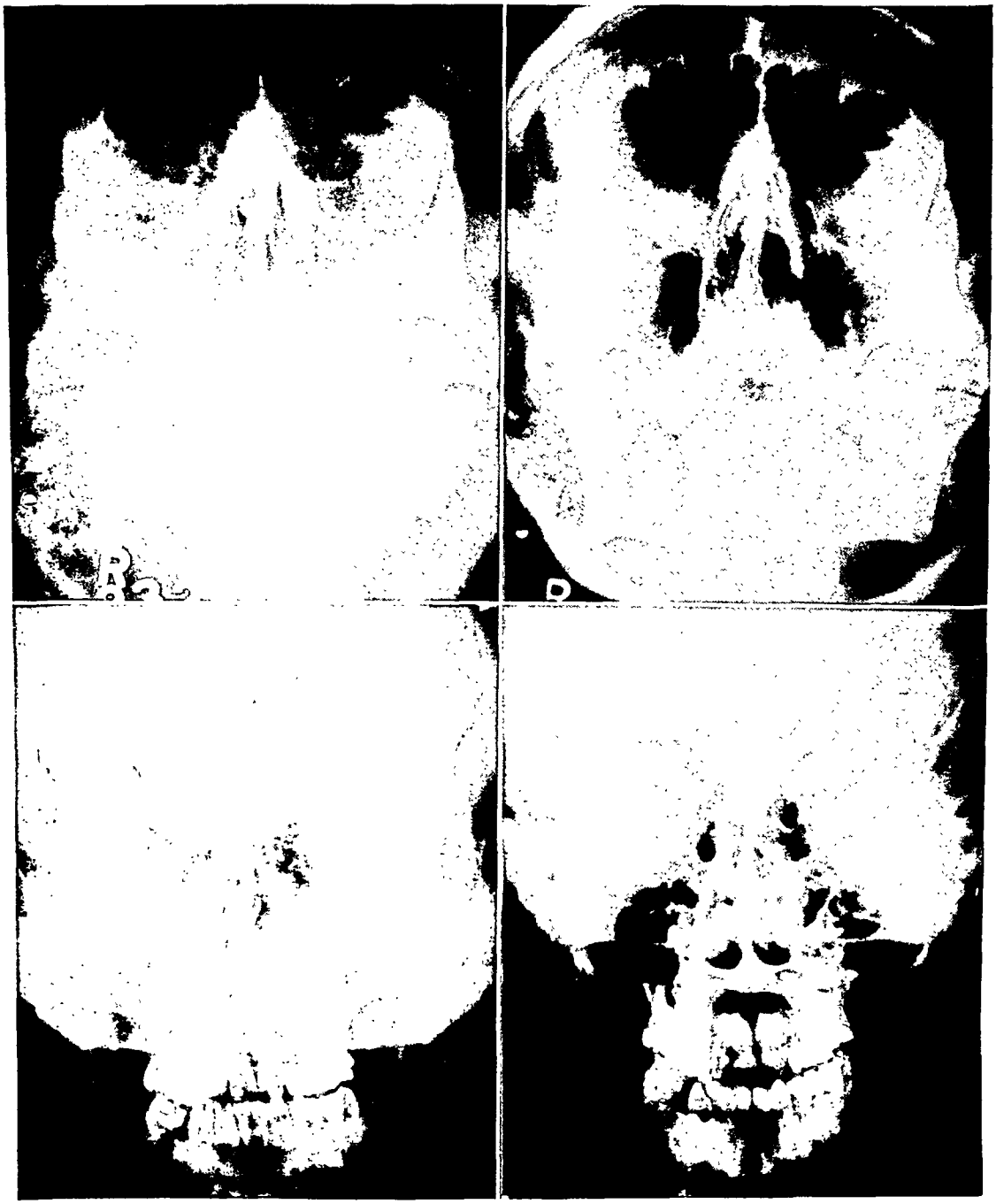
We do not mean to leave the impression that we condemn all surgery of the accessory nasal sinuses. It is obvious that when the normal ostium is completely blocked, surgery is indicated. Patients with symptoms such as pain, headaches, etc., with a rise in temperature, and a swelling of the surrounding tissue, require immediate operation. If operation is unduly delayed in such a case, not only is there a risk of serious complications arising from the extension of the inflammatory process but ultimate healing may be definitely postponed.

mucosa; symptoms reappeared and in ten days there was copious purulent discharge from antra, and severe headaches. Nose drops were discontinued; no relief.

Fig. 2-*C*. Roentgenograph made Jan. 18, 1938, shows increased density in left maxillary antrum. X-ray therapy (300 r).

Fig. 2-*D*. Roentgenograph made April 18, 1938, shows normal radiance of sinuses. Patient began to improve 48 hours after treatment and completely recovered. In response to a questionnaire, he reports that he has remained well since the last treatment.

Perhaps one of the reasons that diseases of the sinuses have been such a problem is that the sinuses, in addition to being a seat of infection themselves, can also be a deli-



A *B*

Fig. 3. Mrs. L. W. M., white, aged 31 years, had sinus trouble "for years." Breathes through her mouth at night, expectorating large amounts of muco-purulent material on arising. Complained of cough, and pain over the antra. Roentgenograph "A" (Dec. 1, 1937) shows increased density of marked degree in both maxillary antra, with right ethmoid cloudy. After x-ray therapy (400 r), patient's headaches were completely relieved; cough and expectoration markedly diminished. Roentgenograph "B" (Jan. 3, 1938) shows all sinuses clear. In response to a questionnaire, patient reports she is now symptom-free for first time in many months.

cate register for changes in other portions of the body of a physiological and pathological nature, as pointed out by Oaks, Merrill, and Oaks (10). Many pathological conditions in the body first show their recognizable symptoms in the membranes of the nose and sinuses.

Many plans of treatment have been devised in an attempt to find a specific therapeutic plan for treating sinus disease, but the horizon has been clouded by failures to the present time. It is true that a few patients will respond to this plan of treatment or that plan of therapy, but who knows but what the patient would have gotten spontaneous relief if left alone. Wenner (20) suggested the use of suprarenal cortex and reported experiments carried out in rabbits with infected sinuses, in which suprarenal cortex was used with good results. But the use of this type of treatment on patients with sinus disease has failed to produce the brilliant results found in experimental animals. Turner and Loew (18) report the use of vitamin A as a method of protection from bacterial invasion of the respiratory tract of rats and suggest vitamin A deficiency as a contributing cause to sinus disease. It is significant that with the widespread use of high vitamin diets and fortification of these diets by synthetic and concentrated vitamin products, if sinus disease is caused by vitamin deficiency, better and more constant results are not obtained. Davison (5) believes this is also true in the allergic type of therapy since the results obtained in treating sinus disease on the basis of allergy are not encouraging except in very carefully selected cases. Reisman (13) believes that when allergy is present it is probably the result of the patient's being sensitized to the proteins in the exudate formed in his own sinuses. If this is true, attention should be focussed upon the sinuses themselves, which are the causative factor, rather than upon the resulting allergy.

The otolaryngologist can relieve a large proportion of the acute or subacute types of sinusitis and a goodly number of the so-called chronic cases, with the use of sound conservative therapeutic procedures. The radiologist enters the picture when, after several treatments, the patient is showing no response.

Radiation therapy can at best relieve only the pathology present in the sinuses. It cannot keep the factors which produce the original disease from causing sinus disease a second or a third time as so frequently happens. Several of our cases have had a recurrence of symptoms, months after treatment. The patients who got relief from the initial treatment also were relieved by subsequent treatments (Figs. 1 and 2).

Osmond (11), in 1923, wrote the first article referring to therapy of the paranasal sinuses by roentgen rays, describing the treatment of acute infections of the antra and frontal sinuses with good results. He stressed that he had not found a method of treatment for all sinus disease.

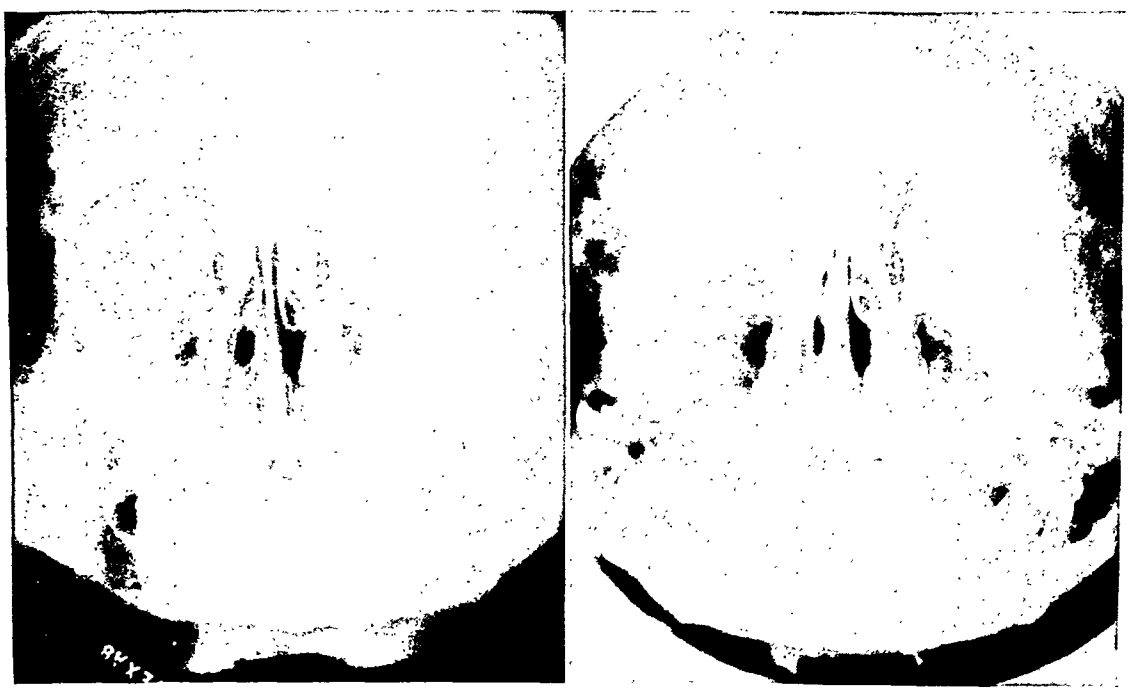
Butler and Woolley, (4), Warren (19), Smith and Nickel (15), Bernheimer and Cutler (2), and others agree that the greatest benefit to be obtained from radiation therapy of the sinuses is in the hyperplastic type of sinusitis in which radiographs show thickened membranes and the patients usually have a thin watery discharge and give a history of colds or chronic cough. The shorter the duration of symptoms, the better the chance of good results, and the greater the possibility of a permanent cure. This coincides completely with our impression of the problem in that our best results have been obtained in cases of this type (Table II).

Since some cases of the chronic type of sinusitis are known to get seemingly miraculous symptomatic relief from radiation therapy, it is of value to give one series of treatments in an attempt to evaluate the response of the individual patient. Several of our patients with chronic changes in the sinuses got prompt relief of all disturbing symptomatology following radiation therapy (Fig. 3).

Fenton and Larsell (6) believe that the benefit of x-ray therapy is due to the early

connection with this type of therapy but in connection with all radiation therapy, it should be carried out only by a thoroughly competent radiologist who realizes the power of the agents which he uses and the posterior, to cross-fire the posterior cells of the ethmoids.

Our present technic closely parallels this, being 200 kv.p., 25 ma., 50 cm. distance, 0.5 mm. copper and 1 mm. aluminum for 10



A *B*

Fig. 4. Mrs. J. R., white, aged 61 years. Asthma, dizziness, and post-nasal drip for one year. Treatment by allergy department, conservative medical and surgical methods, with no relief.

Fig. 4-*A* shows soft-tissue proliferation with blurring of sinus outlines in both antra, particularly the left. X-ray therapy given.

Fig. 4-*B*, made two months later, shows marked improvement in appearance of sinuses. Patient much improved; no asthma since therapy (20 months). Patient gained weight for first time in several years.

terrible effects which can result from their improper use. Irreparable damage and untold suffering can result from the unskillful and careless application of treatment.

Dr. Woolley (22) has given me his latest technic which is not yet published and which is as follows: 200 kv.p., 20 ma., 55 cm. distance, 0.25 mm. copper and 3 mm. aluminum filter, for 10 minutes, which gives approximately 530 r, with back-scattering, through two ports. Port I is an oblique one, through the right side of the face, using a lead mask which exposes only the right maxillary, ethmoid, and frontal area. Port II is the same on the left side. The central rays are directed obliquely minutes, which gives us 400 r, measured in air, without back-scatter, through the single port. The eyes and eyebrows are covered by a lead shield and a lead rubber mask is put over the hair line and just above the chin so that only a 15 × 15 square over the region of the frontal, maxillary, and ethmoid sinuses is exposed.

Smith and Nickel feel that moderate dosage should be used rather than heavy dosage. Rathbone (12) feels that the quality of the radiation is probably of no significance so long as some filtration is used. Rathbone and Smith and Nickel give fractional dosage at intervals for a total of some three to eight treatments. Butler

and Woolley feel that a single full dose gives better results than fractional doses given at intervals. We have used both light dosage and heavy dosage (Table IV). Our experience verifies the findings of Butler and Woolley—that the full single dose gives better results than multiple fractional doses. We now use only the heavy dose method of treatment.

TABLE VI.—RESULTS REPORTED FROM X-RAY THERAPY

Stevens	Cured 80–90%	Improved Not available	Unchanged
Smith and Nickel	60%	78%	12%
Rathbone	57%	28%	15%
Bernheimer	52%	38%	18%
Butler and Woolley	31%	50%	19%
Warren	25%	60%	15%
Martin and Maxfield	18%	35%	47%

Following treatment, our patients have complained of feeling a fullness of the face and increased discharge for from 24 to 72 hours. Following this period, there is a decrease in the amount of the discharge, with a thickening of the discharge itself. If pain is present, it is usually relieved in 72 hours. The patients getting relief during the first 24 to 48 hours following the treatment usually get the best results. The maximum response is usually reached in three to four weeks following therapy. We have found that in most of the cases in which relief has been obtained, a single treatment has sufficed. In cases in which relief has been gained from a single treatment, we have had no hesitancy in repeating the treatment if the symptoms returned and if there had been a sufficient interval of time since our previous treatment. We do not feel justified in continuing the treatment of patients who have received no symptomatic or roentgenographic evidence of relief. Although a few of our cases have been spectacular in the results obtained, unfortunately our percentages of relief and permanent cure are small. We have not been able to confirm the results reported elsewhere. The tabulation of our results are shown in Table VI.

CONCLUSIONS

We feel that although radiation therapy does give relief in a certain percentage of cases, it is not a panacea for all sinus diseases; that it should be used only after conservative therapeutic measures have failed; that it should be used before radical surgery, since it does occasionally produce phenomenal results; that it should be used following all surgery on the sinuses, and that it should be carried out not in competition with but in co-operation with the otolaryngologist and the referring physician.

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PULMONARY INVOLVEMENT IN THE LYMPHOBLASTOMAS: SPECIAL REFERENCE TO ROENTGEN ASPECTS[1]

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THE relative scarcity of articles in the current literature upon pulmonary involvement in connection with the lymphoblastomas would seem to justify the reporting of a series of such cases which the writer has observed within the last few years.

The term "lymphoblastoma" has been used to include a large variety of conditions involving the lymphatic and hematopoietic systems by different authors, Peirce, Jacox, and Hildreth (1) having tabulated 17 separate entities under it. For the purposes of this paper, Hodgkin's disease, lymphosarcoma, and chronic lymphatic leukemia only will be given consideration. The roentgen findings in these conditions are sufficiently alike to warrant grouping them as a unit. Although mediastinal, hilar, and pleuritic lesions are commonly associated with parenchymal involvement, cases in which these structures only appeared to be involved are excluded from this discussion.

As regards incidence, statistics would indicate a rather high percentage of lung involvement. In instances in which these are based on autopsy records, this does not seem surprising inasmuch as the terminal stages of these diseases are undoubtedly associated with more or less generalized dissemination by metastasis or secondary invasion. Many of the changes noted at the postmortem examination are not demonstrable clinically or roentgenologically, which may explain the common impression that such involvement is rare. The high incidence reported by some authors from studies of the living is probably due to the inclusion of lesions limited to the mediastinal and hilar lymph nodes which are relatively common. Falconer and Leonard (2) cite percentages in Hodgkin's disease ranging from 28 to 61 obtained from 125 cases, including 20 of their own, reported by various authors, giving an average of 37.6 per cent. Kirklin and Hefke (3) have reported a 30 per cent incidence in Hodgkin's disease, 23 per cent in lymphosarcoma and six cases of chronic lymphatic leukemia in 48 cases of leukemia. Falconer and Leonard (4) found 36 per cent involvement in 25 cases of lymphosarcoma, and 30 per cent in 30 cases of lymphatic leukemia which they observed. Versi (5) gives a 40 per cent incidence and Moolten (6) states that of 18 most recently autopsied cases in the Mount Sinai Hospital, pulmonary lesions were present in nine and in two others lesions existed within the mucosa of the trachea and bronchi.

The 35 cases which form the basis of this paper, consisting of 24 cases of Hodgkin's disease, six of lymphosarcoma, and five of chronic lymphatic leukemia, are not available for reliable statistics. They constitute about half of all of the cases referred to the author's department for diagnosis or treatment, and they have been diagnosed roentgenologically as probable intrathoracic lymphoblastomas, including mediastinal and hilar involvement. During the same period of observation, our hospital records showed positive biopsy reports of 130 cases. Assuming that all of these cases had roentgen examinations of the chest, it would appear that slightly less than 50 per cent had intrathoracic involvement and in at least 25 per cent pulmonary involvement was present. In 33 of the 35 cases used in this study, the presence of lymphoblastomas was confirmed clinically by biopsy and other examinations; in the other two irradiation

[1] Presented before the Twenty-fourth Annual Meeting of the Radiological Society of North America, at Pittsburgh, Nov. 28–Dec. 2, 1938.

response and other factors left little doubt as to the probable nature of the condition. The lymphoblastomatous nature of the lung changes in most of the cases would seem to have been proved by the fact that they regressed in whole or part after irradiation, as will be discussed briefly in connection with therapy.

Accurate estimates of incidence in the living can be made only if all cases be subjected to roentgen investigation, for this is usually the only certain means of determining pulmonary involvement. This fact has been impressed upon us rather forcefully since we have routinely examined chests roentgenologically in all cases referred to us for irradiation therapy of superficial nodes of lymphoblastomatous origin. Intrapulmonary or other intrathoracic involvement has repeatedly been noted in cases in which there were few or no signs or symptoms suggesting such involvement and no chest examination had been requested.

The pathologic changes found in pulmonary lesions in Hodgkin's disease have been studied in detail and reported by Moolten, and since these changes suggest what the roentgen examination may disclose, it might be well to describe them at length. According to Moolten, the bronchi are conspicuously involved and the entire substance of the walls forms a matrix for the evolution of granulomatous changes. The granuloma is not limited by fibrous tissue sheaths but spreads into the alveolar parenchyma in the manner of a peribronchitis or peribronchial pneumonia. Combinations of granulomatous bronchitis, peribronchitis, lymphadenitis, and perilymphadenitis may produce obliteration of considerable parts of the lung parenchyma, especially around the hilum. There may also be pleurogenous spread which may increase the magnitude of involvement. The process is not confined to lymphoid tissue but may implicate mesenchymatous elements. The granuloma may undergo fibroblastic transformation and in the late stages dense cicatrization may occur. Extensive areas of lung parenchyma may be transformed into solid areas by granulomatous invasion of interstitial tissue between alveoli and the obliteration of them. The bronchial lumen may be affected both as regards its caliber and lining. Polypoid growths may occur in the bronchus and obliterate its lumen, resulting occasionally in atelectasis and bronchiectasis. Abscess formation also has been described. So-called Hodgkin's sarcoma or reticulum-cell lymphosarcoma may be a proliferative phase of the disease.

Part of the parenchymatous changes in the lungs in Hodgkin's disease may be nonspecific, *i.e.*, they may be in the nature of a catarrhal alveolitis or mild irritative pneumonia due to the virus or toxin of the disease rather than to granuloma. There may be an exudative phase with changes general to acute or subacute inflammation.

Search of the available literature has failed to reveal similarly adequate descriptions of the lung pathology associated with lymphosarcoma or chronic lymphatic leukemia. Moolten, in a comparative paragraph, states: "In cases of lymphosarcomatosis involving the lungs, bulky infiltrated alveolar septa may be seen resembling in some respects the analogous condition in Hodgkin's disease." In one of our cases of chronic lymphatic leukemia with pulmonary involvement, the autopsy protocol stated that there were poorly defined nodular areas in both lungs and microscopically there was increase of interstitial tissue in focal areas with dense collections of lymphoid cells. The similarity of roentgen findings encountered in all three conditions would indicate that whatever minute variations occur, the gross pathologic changes resemble each other very closely.

In trying to translate the pathologic changes occurring in the lungs in lymphoblastomas into terms of roentgen images, one is confronted with the usual difficulties encountered in most chronic or slowly developing pulmonary lesions. Site of origin of the disease, stage of development, predominating characteristics of the pathologic changes, and possible com-

plications will all influence the picture presented. A classification for the lesions of Hodgkin's disease suggested by Versi, based on anatomic and roentgenologic features, would seem to serve as an excellent basis for interpretation.

1. Mediastino-bronchial node formation with direct invasion of lung tissue.
 (*a*) By the hilum.
 (*b*) By the medial surface of the lung.
2. Mediastino-bronchial node formation and peribronchial and intrabronchial spread in the lungs.
3. More or less lobar (diffuse) lung infiltration with varying degrees of involvement of the bronchomediastinal lymph nodes.
4. Confluent lobular (isolated circumscribed) focal formation in the lungs with involvement of lymph nodes as above.
5. Miliary dissemination (lymphohematogenous) with involvement of lymph nodes as above (1).

To this might be added occasional atelectasis, bronchiectasis, abscess formation, and a fairly common accompanying fluid accumulation in the pleural cavities as complicating factors.

In going over our cases, we find that all of them conform to one or the other of the above types. Interpretation is comparatively easy if a generalized adenopathy or other signs of lymphoblastoma are present. In the absence of these, in instances in which the patient is first examined because of pulmonary symptoms, the roentgen differentiation may be extremely difficult or even impossible. After other causes have been eliminated as far as possible, an intensive search may reveal inconspicuous enlarged lymph nodes available for biopsy confirmation, or test irradiation may reveal the nature of the condition. Realization of the possibilities of lymphoblastomatous involvements in the lungs and familiarity with the findings they present are prime factors in their recognition. An observation credited to Bernard, and quoted by numerous authors to the effect that a fluid accumulation in the pleural cavity, of unknown origin should suggest the possibility of lymphoblastoma, deserves consideration.

As regards the symptomatology which may be directly ascribed to the pulmonary involvement in the lymphoblastomas, a non-productive cough, dyspnea, and occasionally chest pain or feeling of oppression are common findings and were present in over half of our cases. Other signs of the disease—such as a generalized adenopathy—were present in the great majority of cases and about half of the patients complained of malaise, weakness, or loss of weight. In three instances there was a pronounced pruritus, two patients had specific skin lesions, four gave a history of fever, and nine had fluid in the pleural cavity. No definite conclusions could be drawn of the time the pulmonary involvement bore to the course of the disease, but in several of the cases which were followed from the early stages it occurred as a late or terminal condition. As to age incidence, the youngest patient of the series was nine and the oldest 61, with an average of 35.5 years for the entire group. The Hodgkin's cases averaged 32 years as compared to 42 and 43.5 years for the lymphosarcomas and lymphatic leukemias, respectively. Males and females showed an equal ratio in the Hodgkin's group but there were five males to one female in the lymphosarcoma cases and four to one in the lymphatic leukemias.

In the accurate diagnosis of pulmonary lesions of lymphoblastomatous origin, biopsy findings form the most reliable criteria although regression with irradiation is, in most instances, almost equally conclusive of such involvement. Tumorous masses in the mediastinum, with or without enlarged hilar nodes, and pulmonary infiltrations usually in close proximity to them, are strong presumptive evidence that the condition is of this nature, especially so if the changes are bilateral. Differential diagnosis among the three divisions of this group of diseases is generally admitted to be impossible from the roentgen standpoint, and, although some authors have called attention to individual

variations in the appearance of the lesions or response to irradiation, these variations do not occur with sufficient frequency to render them generally applicable. Kirklin and Hefke have reviewed the conclusions of numerous observers in this respect at some length. The possibility of associated lesions must always be borne in mind, for which reason mediastinal enlargements in suspected cases must be carefully analyzed as possibly being due to substernal goiter, thymoma, paravertebral abscess, or aneurysm. Pulmonary cancers, either primary or metastatic, may on occasion give almost identical pictures and in the absence of other identifying data, test irradiation may be of utmost value. Rarely, pneumoconiosis or chronic pneumonitis may present confusing pictures but usually the history or clinical data permit of easy differentiation. Pulmonary tuberculosis occasionally offers difficulty in differential diagnosis especially in cases in which definite evidences of lymphoblastoma are present and in which the lungs show calcification or other residual changes commonly considered characteristic of tuberculous origin. Lack of response to test irradiation may be of great value in determining the nature of the pathology.

Just as it is almost impossible to correlate the time of appearance of pulmonary involvement with the course of these diseases, so, too, their recognition offers no reliable prognostic indication of the subsequent duration of the disease. Some of our cases were followed for several years after the diagnosis was made (one of them for over five years) and others terminated shortly after its recognition.

As regards the irradiation treatment of pulmonary lesions of lymphoblastomatous origin, it has been stated that these lesions are more radioresistant than superficial nodes; this has not been our experience. Of the 26 cases of our series which had roentgen therapy, 16 showed well marked regression which was repeated in several of them after recurrences. Three of them showed only partial regression. One of these had no biopsy but had generalized adenopathy which responded well to irradiation. (She was observed over nine years, five years intervening between recognition of pulmonary involvement and fatal termination with pleuritic complications.) One other of these three had questionable pathologic findings after two biopsies. Four of the patients showed practically no change, and in two of these the roentgen findings, although consistent with Hodgkin's disease which according to biopsy report was present, showed calcification suggestive of an old tuberculous lesion. One of these four, whose biopsy diagnosis was lymphatic leukemia, had an interstitial fibrosis which may have been the result of an industrial hazard. The fourth case, also one of Hodgkin's disease, had bone lesions as primary manifestations and ran a rather rapid course. Two cases in the Hodgkin's group showed progressive changes; one of these also had marked bone changes and succumbed shortly after he first came under observation. The other was diagnosed as Hodgkin's sarcoma. One other case included in this series has been similarly classified but roentgen therapy given him has been of too recent date to permit of drawing conclusions as to results. Four of the six cases of lymphosarcoma received irradiation and all of them showed regression. Of the five cases diagnosed as chronic lymphatic leukemia, three received roentgen therapy but only one showed well defined regression of the pulmonary lesion.

A study of the series of cases which forms the basis of this report has led to the following conclusions:

1. Pulmonary involvement of lymphoblastomatous origin is relatively common.
2. Roentgen findings produced by it are sufficiently characteristic to be suggestive of the condition.
3. Routine chest examination of patients known to have lesions apparently limited to the superficial nodes would result in its more frequent recognition.
4. Test irradiation of suspicious lesions is a valuable aid in the differential diagnosis.

I want to acknowledge my appreciation and thanks to Dr. T. J. Wachowski and Dr. J. W. Grossman, of the Department of Roentgenology, for their aid in collecting and preparing the material used in this paper.

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DISCUSSION

GEORGE W. GRIER, M.D. (Pittsburgh, Pa.): I am sorry that Dr. Hartung did not have time to read all of his paper. If he had, I am sure he would have impressed you all, as he did me, by the thoroughness and care with which he has gone into this subject. It is a fine example of what we all should do in studying our cases.

In my own mind I would be inclined to separate lymphosarcoma from leukemia and Hodgkin's disease because I am always expecting to find metastasis or involvement in the lung in lymphosarcoma, but I must admit that I had not been so prone to consider the probabilities of infiltration of the lung in the other two conditions and I was rather surprised to read his figures regarding the incidence of this condition or this complication.

I had been inclined to look on the invasion of the lung, especially in Hodgkin's disease, as a very, very late involvement and to be seen only in the terminal stages. I have been forcefully impressed, by this paper of Dr. Hartung, with the desirability of studying our cases more thoroughly. When we get a cancer of the breast to treat, the first thing we do is to make a film of the chest, and it is obvious to me, after hearing Dr. Hartung, that that is the first thing we ought to do in these cases of Hodgkin's disease and leukemia.

The thing that has impressed me most in this presentation is the high incidence of involvement into the lung; I was particularly glad to hear this paper because the essayist gave proof of the nature of these involvements as examined at autopsy. I have speculated many times as to these involvements, particularly that of Hodgkin's disease—whether they were actually invasions of the lung with what we might call "Hodgkin's tissue," whether they were simply involvement of the lymphoid material in the lung, or perhaps simply cases of atelectasis. Dr. Hartung has analyzed these in his paper and mentions that all these conditions exist. I was glad to learn that the pathologists have found there is definite invasion of the lung by granulomatous tissue—that has cleared the matter up in my mind.

I must admit that I, and possibly others, have been inclined to pass over this question of invasion into the lung as not having much significance, but, as a matter of fact, we can easily see that it does have significance, because these involvements will need treatment just as well as the glands. I am referring entirely to the invasions into the lung and not to the glands in the mediastinum or in the hilus. Of course we all expect them. In Dr. Hartung's paper he mentioned that it was generally considered that these lesions were quite radioresistant, but I believe in his talk here he said that he considered them of the same sensitivity to radiation as the superficial lesion. I am inclined to that opinion.

STEWART HARRISON, M.D. (London): The illuminating paper of Dr. Hartung and the discussion by Dr. Grier on this important subject of mediastinal tumors prompt me to report an experiment that I made some four years ago in Zürich under Professor Schinz and have confirmed recently.

It fell to my lot to collect the material on mediastinal tumors in the Canton Hospital during the twenty preceding years and after excluding, as far as was clinically possible, the type of Hodgkin's disease, the tuberculous type, the thymomas, there remained some 36 cases that fell in the category of tumors of the lymphoblastomatous series.

These tumors were accompanied by the usual clinical symptoms, of course, of a mediastinal tumor, and to a varying degree with blood changes indicating lymphatic leukemia. The blood changes ranged from a maximum of a frank, serious leukemia through the leukemic phases in which the type of lymphocytes present was indicative of leukemia but there was no increase in their number, to cases in which there was absolutely no alteration of the blood picture of any kind.

The point I wish to make is that there were six of this latter kind—tumors of the mediastinum with dyspnea, maybe cough, and no blood changes of any kind, no evidence of Hodgkin's disease, no relapsing temperatures (and they were called lymphosarcoma)—from that, by very small gradations, to the frank lymphatic leukemia tumors.

All of these patients, except two or three who were too moribund, were subjected to radiation of their mediastinal tumors. All with a fairly marked lymphatic leukemia improved, only to relapse, and are now dead following the course of lymphatic leukemia, only too familiar to us all.

Of the six cases in which there were no blood changes of any kind and which were referred to as "lymphosarcoma"—whether or not that is a distinct entity I was unable to conclude—three showed no response at all. They rapidly went downhill and within a month or so were dead.

Of the others, three cleared up, have never relapsed, and remain clear at the present time. The only three in this group of 36 that have cleared up were cases in which there were no blood changes at all and in which the diagnosis was put down without biopsy (which was unobtainable) as lymphosarcoma. I would be glad to hear from Dr. Hartung as to whether or not he has made any such experiments—that tumors of the mediastinum unaccompanied by blood changes give the most favorable distant results after irradiation of a fairly heavy character.

ADOLPH HARTUNG, M.D. (*closing*): I have little to add to my paper. I want to thank Dr. Grier for his kind discussion.

In regard to the remarks of the last speaker, as far as prognosis is concerned, of course the prognosis in practically all cases of lymphoblastoma is bad. Some of the cases that we have observed, as shown here to-day, have been followed for as long a period as nine years and in one of them, in which pulmonary pathology was present, the patient survived for five years after the involvement was noted.

I can give no figures in regard to the blood findings in the cases of lymphatic leukemia, but the last case shown certainly showed no regression under irradiation and most of the other cases that we treated responded in the same way.

YEAST GROWTH: A POSSIBLE TEST ORGANISM FOR X-RADIATION[1]

By OSCAR W. RICHARDS, PH.D., *Buffalo, New York*

From the Research Department of the Spencer Lens Company and the Osborn Zoölogical Laboratory, Yale University

LARGE numbers of yeast may be grown under closely reproducible conditions with little special equipment, because of the accumulated knowledge about the growth of this organism. Even though yeasts were known to be resistant to the effects of x-radiation, the other advantages of this organism suggested that it might be useful as another dosimeter. Preliminary experiments during the Summer of 1931, at the Marine Biological Laboratory, showed that the population growth was arrested for a time by x-radiation, then the culture apparently recovered and continued to grow at the original rate. The period of arrested growth varied with the amount of radiation.

I

The resistance of yeast to x-radiation may account for a considerable part of the lack of agreement in the published investigations. In some of the publications on this subject it is not clear which species of yeast was used or that the cells were grown under optimum conditions. Fresh baker's yeast was found to be little affected by x-radiation (Wels, 1924). Nadson and Zolkevic (1923) showed that potassium chloride had a protective effect when yeast colonies were irradiated on beerwort agar, but that sodium chloride did not. Nadson (1925) postulated that x-radiation stimulated the rate of living of organisms. Stimulation of *S. ellipsoideus* by small doses of radiation is denied by Lacassagne and Holweck (1930). Old cells were more sensitive than young cells, and a change in temperature did not change sensitivity. Burger (1931) reports 100 per cent killing in ten minutes with 35 kv., 10 ma. at 3 cm. with radiation of about 0.35 Å. Lower voltages had no appreciable effect. The effects of x-radiation and ultra-violet radiation are compared by Nadson and Stern (1931), who found that the young cells are more sensitive: the vacuole contour changes first, then the metachromatin coagulates and the cell dies. Ultra-violet radiation caused more rapid death. A curious oligodynamic action was also described by these investigators.

Wyckoff and Luyet (1931) irradiated old cultures of a pure strain of *S. cerevisiæ* and found that the survival curves were of the type representing a multiple-hit-to-kill hypothesis. Many cells divided once, and in other cases separation failed and giant fusion cells were formed. Imshenetskir (1932) found 40 per cent of a 24-hour culture of *S. cerevisiæ* killed by 10 H.E.D. and that certain dyes gave a photodynamic sensitization to the roentgen rays. Holweck and Lacassagne (1930) summarized their findings on the yeast *S. ellipsoideus.*

Irradiation of yeast, after the beginning of fermentation, was found by Schneider (1925) to reduce the carbon dioxide production. Zeller (1926) stated that x-radiation produced a latent change in the cell substance, shown by increased sensitivity, but had no effect on the fermentation enzyme within the cell. An increase in the rate of carbon dioxide production was observed by Gronchi (1931) to be proportional to the intensity and was inversely proportional to the wave length. Each radiation was adjusted to 45 minutes with from 300 to 2,000 r and wave lengths of 0.55, 0.37, and 0.16 Å. Fardon and his co-workers (1936) stated that a substance liberated into the

[1] Through the kindness of Dr. G. Failla and a grant from the Atypical Growth Study Unit of Yale University it was possible to test the effects of radiation on yeast in the Biophysics Laboratory of Memorial Hospital, New York City. The hospital staff attended to the problems of radiation, which left the author free to measure the growth of the yeast. I am greatly indebted to Dr. Paul S. Henshaw for arranging the work in the laboratory so efficiently.

medium might account for the effect of the radiation.

Genetic variation producing new strains was believed by Nadson and Philippov (1928) to result from x-radiation of yeast.

Washed beer yeasts were irradiated by Wels and Osann (1925) and the growth of the populations was checked by 35, 38, 63, 77, and 88 per cent, respectively, after one, two, four, four, and eight hours of radiation (one hour equalled 12 H.E.D.). Neither the oxygen uptake nor the carbon dioxide production was affected, as measured by the Warburg technic. Large amounts of radiation are believed to produce an immediate death of the cell, while somewhat less radiation may cause a delayed death after one or more divisions of the cell (Lacassagne and Holweck, 1930; Lacassagne, 1930).

Fardon, Norris, and Ruddy (1936) found a variation in the effects of x-radiation and ultra-violet on the respiration of yeast. This was not due to the age of the yeast or the *p*H of the culture medium. They believe that the injured cells secrete a substance, or that the radiation activates a substance which stimulates the respiration of the remaining yeast.

The problems of radiation effects and the types of lesions have been summarized by Lacassagne (1934) and by Crowther (1938).

Consequently, the effect produced may appear differently, depending on how and when the growth of the yeast was measured and the next step in the analysis should be toward standardized procedures.

II

The rate of growth of a yeast population in an adequate and effectively constant environment is constant (Richards, 1928). When the population starts in a given amount of culture medium, the environment becomes progressively unfavorable and ultimately the growth is limited because of the exhaustion of food and the accumulation of injurious waste products. These changes have been measured for the pure strain of *Saccharomyces cerevisiæ* which Hansen obtained from a single-cell isolation (Richards, 1932, 1934).[2] While several criteria of growth are necessary to obtain a complete picture (Richards, 1933), one or more may be used in a given experiment, provided their limitations are kept in mind.

The most generally used criterion is the number of cells present in a given volume, often $^1/_{250}$ cu. mm., at a given time, obtained by averaging the counts of several samples made with an hemocytometer (16 small squares). The errors of the hemocytometer method for blood counting have been discussed by Berkson, Magath, and Hurn (1935). With care, the overall error may be reduced to about 4 per cent for yeast populations. Unless information is available showing the proportion of the cells of various sizes from recently formed buds to fully grown cells, the cell count does not give complete information as to the amount of yeast substance present. Other criteria have been used and the interrelations of some of these have been described (Richards, 1934) and a more complete analysis will be published later.

The optical density of a suspension of yeast cells may be readily measured by a photo-electric nephelometer (Richards and Jahn, 1933). When I is the amount of light transmitted to the photo-electric cell by the medium and I_t the amount transmitted by a population of cells in the medium at time t, then the optical density,

$$D = \log I_t - \log I = \log (I_t/I).$$

With the Richards and Jahn instrument, the number of micro-amperes generated by the light may be used as a direct measure of I. The nephelometric criterion is dependent on the number of cells present, the proportion of cells of each of the various sizes, and the condition of the cell protoplasm and wall. The values of D are a sort of inclusive measure of the yeast

[2] Used by Richards since 1928, and furnished to others. Some seven years after the isolation, a transfer sent to Dr. A. E. Navez duplicated the author's previous growth curves, which indicates the stability and reproducibility of the growth when the conditions are comparable.

growth, which is difficult to correlate directly with a single specific criterion such as cell number. The measurements are slightly more precise than counting, when the greater part of the population is within the light beam.

The instrument consists of a 100-watt projection bulb mounted in a lamp house to direct the light through a block onto a suitably screened photo-electric cell. The block is drilled at right-angles to the beam and so made that a test tube, containing a yeast culture, can be replaced in the instrument in the same position. Recent improvements of the original instrument are the addition of a ballast tube to lessen the fluctuations of the 110 v. lighting circuit, the use of a G.E. Blocking Layer type of photo-electric cell and a Weston No. 600 micro-ammeter with a mirror and a knife-edged pointer.

The yeasts are grown in Williams' medium, consisting of 20 gr. cane sugar, 3 gr. ammonium sulphate, 2 gr. potassium dihydrogen phosphate, 1.5 gr. asparagine, 0.25 gr. each of anhyd. calcium chloride and magnesium sulphate, and 1 liter of distilled water. Ten milliliters were placed in 2 × 15 cm. Pyrex test tubes and sterilized in an autoclave for 15 minutes at 12 pounds. This amount of heating does not affect the sugar, but repeated sterilization should be avoided. It is desirable to test the test tubes with culture medium and reject those which do not give the same reading on the photo-electric nephelometer. The maximum crop or yield, in 10 milliliters of medium, will depend somewhat on the chemicals used, hence it is advisable to start with sufficient stocks so that all the experiments of a given series will be made with the same chemicals. Merck's reagent grade chemicals were used in the experiments reported here except that the asparagine was Merck C.P. and the sugar was "Jack Frost." Any good table sugar may be used, although different brands of sugar will give some variation in the yields of yeast. The variations in growth, due to impurities present in the chemicals, are more or less well known. When it is necessary to change chemicals, a careful comparison should be made between the new and the old.

The yeast used should be a pure strain from a single-cell isolation, preferably of a well-known culture. The yeast used in these experiments is available as No. 4360 of the American Type Culture Collection. The stock strain may be kept on Difco malt extract agar or wort agar slants, by transferring about every four months. The transfers and inoculations should be made with the usual precautions used in bacteriology to avoid contamination. The yeast should be grown in a constant-temperature incubator at about 28° C. to avoid any special effects of higher temperatures (Richards, 1928).

The seed yeast used in an experiment should not be older than within the logarithmic phase of growth, in order to prevent the confusion of any lag period.

The growth then will continue for a period of time at a constant rate which is demonstrated by a straight line when the logarithm of the growth is plotted against time or when arithlog plotting paper is used. This period is called the logarithmic period and is readily duplicated and experiments made with yeast in this phase of growth are directly comparable. After the phase of constant rate of growth, the rate gradually decreases and an equilibrium number is attained by the population. A second smaller cycle of growth may follow the first. As the equilibrium is reached, the distribution of cell sizes changes from a preponderance of small cells to larger cells. In old populations the cells gradually change into resistant cells. These changes have been described for this strain of yeast (Richards, 1932). Old cells should not be used in experimental work because of the difficulty of determining their exact physiologic condition, and of duplicating the same population from experiment to experiment.[3]

[3] Precautions necessary for the use of yeast growth when testing the effects of various influences on it have been stated and discussed by Richards and Taylor (1932).

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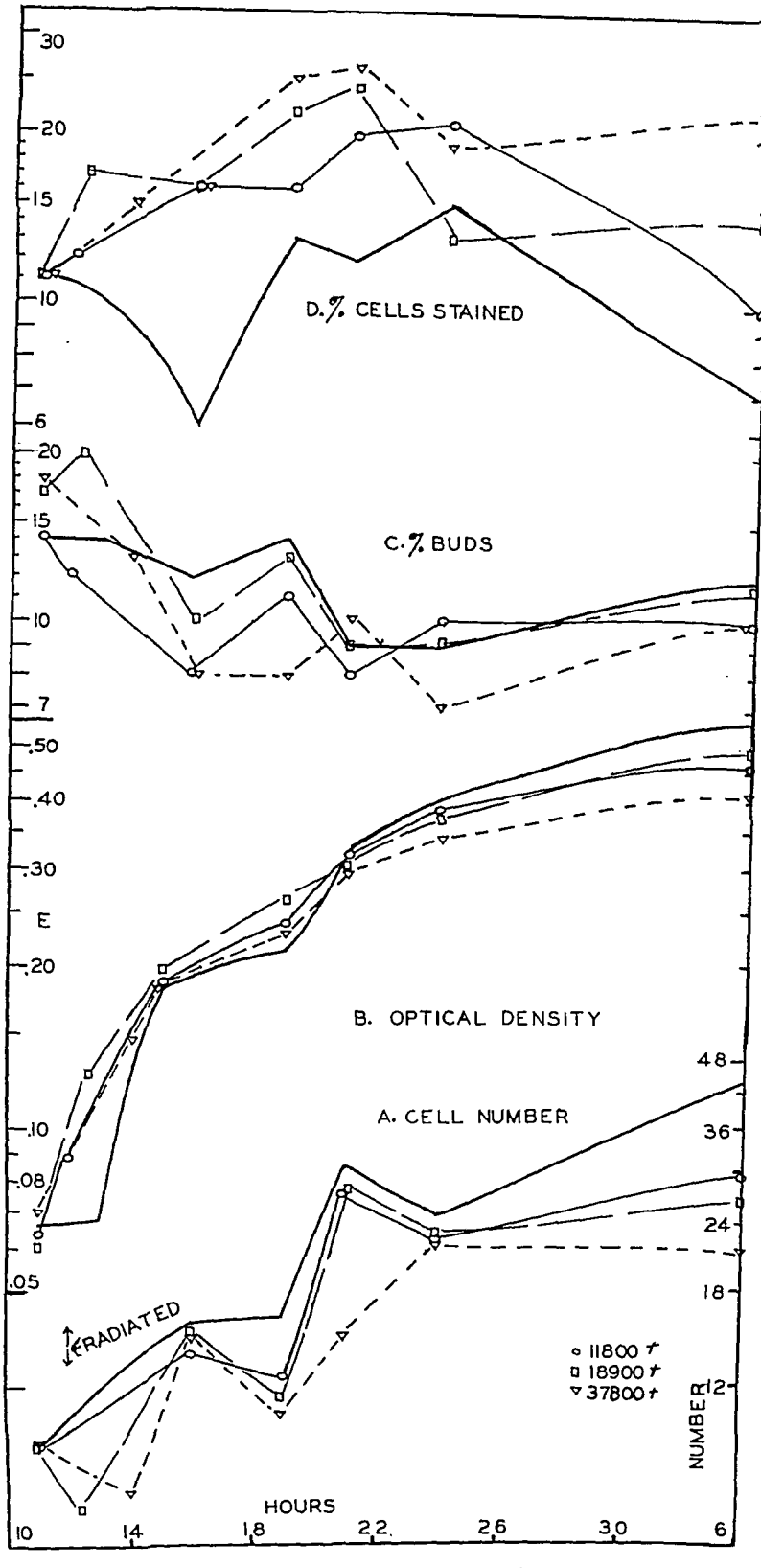


Fig. 1. *A*, Increase in number of yeast cells; *B*, Increase in optical density; *C*, Percentage of buds; *D*, Percentage of cells staining with methylene blue (*cf.* text). Smooth line is unirradiated control.

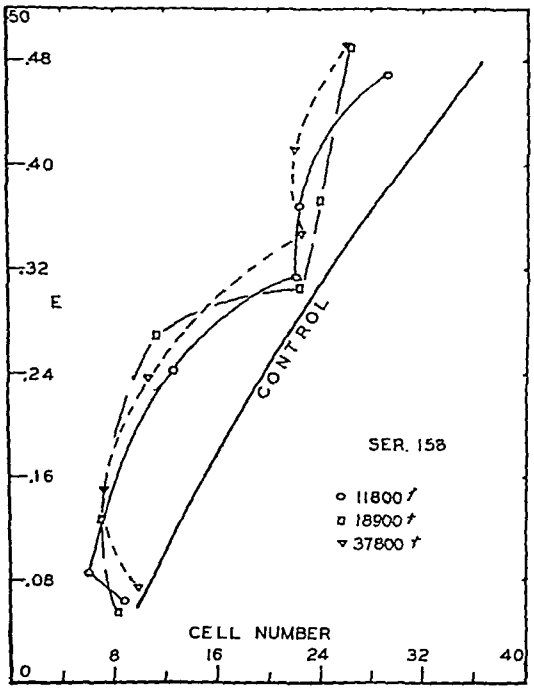


Fig. 2. The relation between number of yeast cells present per unit volume and the optical density of the population measured with the nephelometer.

nearly the same level of equilibrium as the controls at the end of the growth cycle. Two criteria of the effect of the radiation could well be the length of time following radiation until the relative rate of growth became the same as the control population, and the amount of reduction in the population size at a given time after the irradiation. With so few experiments, it is premature to state these values, but further experimentation should make possible the determination of the constants for different amounts of radiation.

The optical density, measured by the nephelometer (Fig. 1-*B*), shows that there was a greater density in the irradiated cultures for about nine hours following exposure and then the density became less than that of the controls. During the logarithmic period, the distribution of cells of different sizes present in the cultures is the same. Less cells are present in the irradiated cultures and, as will be shown later, less buds are present; therefore, the x-radiation must make the content of the cells more dense so that less light passes through them than through the control cultures. The populations receiving more than 15,000 r show an optical density less than that of the control populations.

The optical density and cell number are compared in Figure 2. The control culture shows a regular increase in the optical density corresponding to the increased number of cells in the population. The optical density of the irradiated cells is greater and reaches a maximum at about nine hours after exposure to the x-rays. The difference decreases, then increases, and at the end of the growth cycle becomes about the same as the controls for the same number of cells in the populations.

The percentage of buds is a measure of the birth rate and is plotted in Figure 1-*C*. The percentage of buds decreases markedly after irradiation and reaches about the same value as that of the controls by about twenty-five hours following irradiation. The effects showed considerable variation within and between the experiments, but in no case showed any stimulation of bud formation. The decrease in buds accounts for part of the lesser number of cells present in the populations after irradiation.

There is, unfortunately, no satisfactory easily performed test for dead cells. Culture of the individual cells is the only certain method of showing that they are or are not able to reproduce, although they may still live. Break-up of the cells apparently takes place shortly after death, and little evidence of them remains in the medium as the materials liberated are used in part by the remaining viable cells. Cells injured or moribund become deeply stained by methylene blue when a drop of a 0.5 per cent solution is added to four drops of the culture. The validity of the staining criterion is questioned,[4] but it remains a generally accepted means of evaluating the healthiness of the cells.

The percentage of stained cells is shown in Figure 1-*D*. More and more of the ir-

[4] A discussion and literature references to the staining of injured cells is given by Richards (1932). Dr. P.S. Henshaw's experiments, subsequent to the author's, indicate that methylene blue staining may not be as useful for the study of x-radiation injury.

radiated cells are stained for about nine hours following the exposure to x-rays, then the number decreases and ultimately approaches the level of the control populations at the end of the growth cycle. The changes in the protoplasm, which result in staining, are also probably responsible for the increased optical density. The minimum difference between the optical density of the controls and the irradiated cells (Fig. 2) occurs at the time of maximum staining.

IV

Neither the optical density (nephelometric) criterion of growth nor the increase in the numbers of cells present in a unit volume of culture medium alone is adequate to evaluate the effect of x-rays on yeast growth. The information of each assists in the interpretation of the observations. A complete study should include also the determination of the volume of cells present and the numbers of cells of different sizes.

The counts were made at intervals of about three hours after the exposure to the x-radiation until late evening, and then three times a day until the population equilibrium was reached following the second cycle of growth at about one hundred hours. The data given in Figures 1 and 2 were taken at three-hour intervals from carefully drawn, but not smoothed, plots of the observed growth. Much of the irregularity in the growth was due to the cooling of the cultures while they were out of the incubator during the withdrawal of the sample for the count.

If the injured cells divided once and then died, the curves showing growth on the arithlog plots would be moved over and then closely parallel that of the control populations. This could not have happened to any great percentage of the yeasts in these experiments. The staining criterion shows that a large proportion of the injured cells were buds. This double type of killing would account for the lack of systematic variation in the plots of Figure 1.

The effects with time on a growing population will be different from those on a population suspended in saline or other medium which will not support growth. Cells growing at a potentially unlimited constant rate will probably behave differently from old cells recovering from the adverse effects of an unfavorable environment. These factors must be considered and controlled in studies on survival.

The staining of the cells injured, and the lack of it by the normal cells, suggested that this, coupled with the reduction of the methylene blue to methylene white by the yeast populations, might be another measure of the effect of x-radiation on growing yeast cultures. A given amount of the dye was added to the irradiated and control cultures and the amount of reduction measured with the nephelometer at the end of 25 minutes. The following differences in the reduction of the dye were obtained with two series: 4,000 r, 19 units; 12,000 r, 22 units; 18,000 r, 18 units of optical density. Roentgen rays do affect methylene blue when the exposure is direct (Stenström and Lohmann, 1934; Stenström and Street, 1935).

V

To compare the rates of growth the relative rates (dy/ydt) were computed for the cultures at the same intervals of time[5] and comparisons made by subtracting the relative rate of each irradiated culture from that of the control at the same time interval. The differences are shown in Figure 3. Values to the left of the central zero line show increased relative growth of the irradiated culture and those to the right show retardation in the growth of the irradiated cultures in comparison with the relative rate of growth of the control populations. The smooth lines are the values for number of cells present and the dotted lines show the data from the nephelometric criterion. Much irregularity is present, as is to be expected from the small number of experiments permitted while covering this range of dosage in the available time. The

[5] The methods and calculations are described by Richards and Kavanagh (1937).

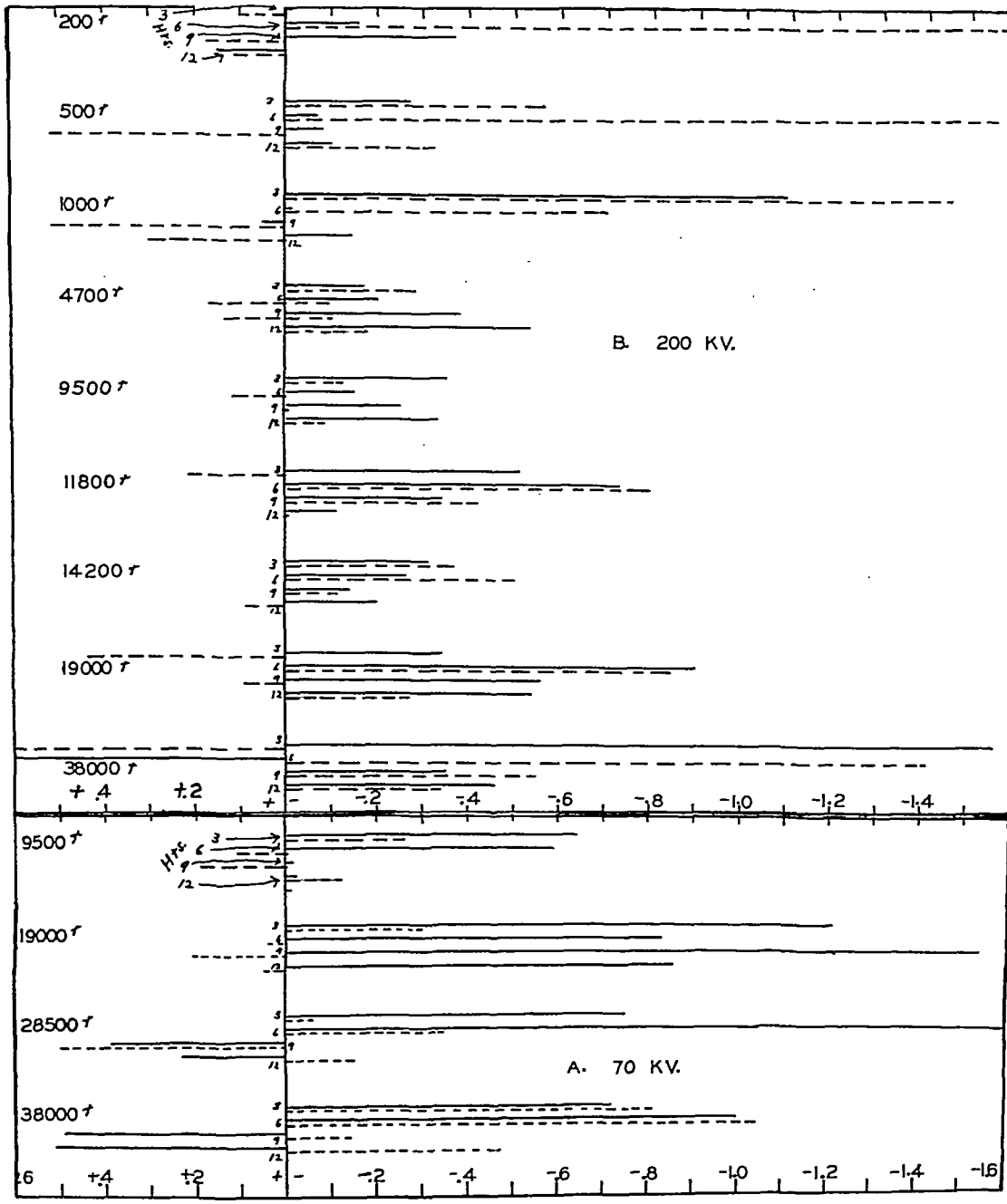


Fig. 3. The changes in relative growth rates following x-radiation. Solid lines, cell number; broken lines, optical density. The first two lines of each group show the difference between the relative growth rates of the irradiated and the control population at three hours after irradiation, the second at six hours after irradiation, etc.

200 kv. radiation gave little evidence of stimulation and there is very slightly more shown for the experiments with the 70 kv. radiation. Differences of less than 0.10 are probably not significant. Recovery from the effects of radiation occurs earlier with the two higher dosages of the 70 kv. radiation, which suggests that the cells are killed and the survivors less injured than appears from the 200 kv. irradiated populations. The lower dosages and the higher ones at 200 kv. are more effective in

retarding the growth rate of the yeast populations.

The survey experiments here reported show that several criteria of growth must be used simultaneously to analyze the effect of x-radiation on the growth of yeast. With properly replicated experiments, with a pure strain of yeast grown under appropriately controlled conditions, it is believed that yeast will be a useful test organism for the analysis of the effects of larger amounts of radiation and the clinical use of large amounts of radiation suggest that development of the methods outlined will be of value in experimental biology and medicine.[6]

SUMMARY

Populations of a pure strain of *S. cerevisiæ* were radiated with from 9,500 to 28,500 r at 70 kv., and with from 200 to 38,000 r at 200 kv. under carefully controlled, established conditions for the growth of yeast. The increase in number of cells per unit volume of culture is retarded by the x-rays. The growth measured with a nephelometer showed increased optical density for dosages up to 15,000 r, and decreased optical density for greater dosages. Recovery gradually takes place, and the irradiated cultures reach about the same equilibrium growth at the end of the growth cycles as do the controls. The percentage of buds and the percentage of cells stained with methylene blue are given. While a single criterion of growth may be useful, the analysis of the effect of x-radiation on yeast growth requires simultaneous information from several different methods for the measurement of yeast growth.

[6] The methods developed by Bliss (1935–1938) should assist in studies on the effect of radiation when sufficient data are available.

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THE INCIDENCE AND SIGNIFICANCE OF APICAL LESIONS IN PULMONARY TUBERCULOSIS: A PATHOLOGIC AND ROENTGENOLOGIC STUDY[1,2]

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ALTHOUGH the various phases of pulmonary tuberculosis, including its pathologic and roentgenologic aspects, have for many years been favorite subjects of investigation, scarring at the apex of one or both lungs such as is so often present in lungs examined at necropsy, has received relatively little attention in the literature. That the lesion is tuberculous scarcely can be doubted, for Opie (1) observed evidence of tuberculosis in many cases, and Baldwin (2), Petroff (3), and Gardner (4) have stated that living bacilli of tuberculosis have been found in the lesion.

Examination for this lesion has been a routine procedure in the Department of Pathologic Anatomy of the Mayo Clinic for several years. The lesion is almost always at the extreme apex of one or both lungs (Fig. 1). It is seen as a whitish, opaque scar that stands out in contrast to the normal color of the apical visceral pleura surrounding the lesion. The portion of the apex covered by the scar varies considerably. It may be found, in minimal cases, as just a slight puckering and opacity or whitening of the visceral pleura. In extreme cases, the scarring may cover the entire apex, forming a cap. There are also many variations as to the shape it may take. It is interesting to note that it is never found where the rib has encroached on the lung. The thickness of the scar probably varies more than the surface extent. On sectioning the lesion, the thickness will vary from 1 mm. to 8 or 10 mm. (Fig. 2).

No evidence of a similar lesion of the parietal apical pleura adjoining this has been found except in cases in which adhesions joined the two layers of pleura at the point of scarring. Such adhesions were found in very few of the cases studied. On palpation of this area of the apex with the finger tips, the definite resistance of the scar compared to the surrounding normal apical pleura can at once be appreciated. In the less severely affected cases, the parenchyma of the lung directly beneath the scar is uninvolved; occasionally one will find an emphysematous cavity 2 or 3 mm. in diameter or multiple smaller ones.

On examining these apical scars with the aid of the microscope one sees a picture of complete fibrosis (Fig. 3). In many of the sections the connective tissue has become hyalinized. As a rule, there is no evidence of proliferation of blood vessels. In many of the sections in which there has been a general anthracosis of the lung, coal pigment will be found deposited in clumps between the connective tissue fibrils. Evidence that silicon might be interspersed with the coal dust is lacking when these areas are viewed through a polarizing microscope. Tubercle formation, giant and epitheloid cells are seldom seen.

The lesion is manifested roentgenographically as an opacity at the extreme apex of the lung (Fig. 4), its density varying considerably according to the extent of the scarring. It is seldom an obvious radiographic image, and to be sure that one is dealing with an apical scar the films should be stereoscopic and should be viewed in a stereoscopic viewbox. The lesion occurs almost always within the arc of the first rib and in the posterolateral quadrant

[1] Adapted from a thesis submitted by Dr. Addington to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Roentgenology.

[2] Accepted for publication in October, 1939.

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of the apical cupola (Figs. 5 and 6). The visceral margin is sharply defined and is usually slightly concave toward the lung-

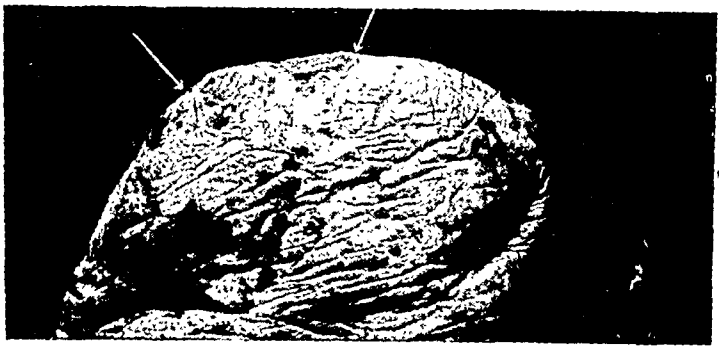


Fig. 1.

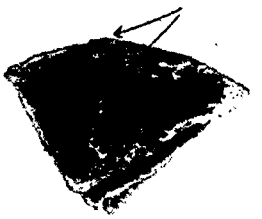


Fig. 2.

Fig. 1. Actual photograph of an excised lung with apical scarring.
Fig. 2. Thickness of apical scar as shown by actual photograph of excised lung. A section has been cut down through the middle of the scar.

field. The density may be so slight that only the line of the visceral margin is seen to suggest its presence, and the average density is rather light. Occasionally evidence of calcium can be seen in the shadow. The opacity can never be seen to cross the first rib. Stereoscopically it appears to be just inside the first rib and is distinctly not a bony structure.

Naegeli (5), in 1900, was the first to mention the existence of this type of tuberculous lesion. He made the statement that it could be found in practically 90 per cent of persons who had lived in cities on the continent of Europe, and who had died of causes other than tuberculosis. Opie (1) was the first investigator in this country to draw attention to apical scarring. He found it most often in patients in the later decades of life and believed it could be found in one out of every five or six adults dying from causes other than tuberculosis. He also found a great many of these lesions to be active, and at the same time, the primary complex to be calcified. Consequently, he believed this lesion to be a secondary type of infection derived from an exogenous source. During the World War he had the opportunity to perform necropsies on a number of British soldiers killed in action and he found evidence of healed tuberculosis of mesenteric nodes in many of these cases; he also found apical scarring with but a few having the calcified Ghon complex.

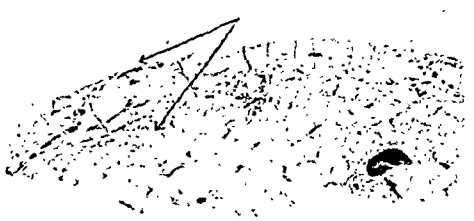


Fig. 3. Photomicrograph of a section through apical scar (× 8)

Van Zwaluwenburg and Grabfield (6), in 1921, investigated scarring at the apex from both the pathologic and the roentgenologic standpoints. They were quite well convinced that the lesion was closely associated with previous tuberculous infection of the tonsils or of lymph nodes in the cervical region. They believed that the tuberculosis bacilli gained access to the apical pleura directly by way of the lymphatic chain of the cervical region. They determined by roentgenogram that 93 per cent of the patients with tuberculosis of the tonsils had evidence of apical scarring and that 59 per cent of the patients with apical scarring had tuberculosis of the

cervical lymph nodes. In the large majority of patients on whom necropsy was performed they found evidence of adhesions between the visceral and parietal pleura.

On the other hand, Beitzke (7), in 1906, studying the routes by which the tuberculosis bacilli could enter the lung parenchyma, found no connection between the lymphatics of the neck and the cupola of the pleural cavity.

Grober (8), working along the same line of thought as Van Zwaluwenburg and Grabfield (6), injected the pharynx of dogs with India ink. The dogs were then killed (at intervals) and the lymphatics of the neck and apical cupola were dissected out to determine the dissemination of the ink. He found that it had been carried down the cervical lymphatics directly into the pleura of the apex. In some instances he found traces of the ink in the parenchyma of the lung beneath the apical pleura. From this study, he concurred with Van Zwaluwenburg and Grabfield that apical scarring was brought about in this manner and that this was possibly the route of infection in many cases of pulmonary tuberculosis. He postulated that the apical pleura offers a good deal of resistance to the bacilli and thus prevents, in the majority of cases, a parenchymal tuberculous infection. This, he thought, was the reason that apical scarring was such a benign lesion.

There is some evidence in the literature that the lesion may be the focus of an adult type of tuberculous infection. Fishberg, (9, 10), Allison and Medelman (11), and Pohle and his co-workers (12) have cited cases in which this has occurred. Allison and Medelman expressed the opinion that, in children, when the apical lesion is the focus, it causes a fulminating type of parenchymal tuberculosis.

The reported incidence of apical scarring in stereograms of the thorax varies a great deal. Pohle and his co-workers (12), of the University of Wisconsin, found evidence of scarring in 1.4 per cent of 2,072 students who had roentgenographic examinations of the thorax. Van Zwaluwenburg and Wickett (13) found that 10 per cent of patients registering at the University of Michigan Hospital had evidence of scarring at the apex. Andrus (14) found scarring in 27 per cent of a series of 1,085 stereoscopic films of normal adults.



Fig. 4. Roentgenogram of an excised lung with apical scar. The scar is seen at the extreme apex on its medial margin.

Few who have studied this type of tuberculous lesion have ventured to explain the mechanism by which the scarring occurs. As one explanation, Opie and McPhedran (15) have expressed the opinion that this lesion is a secondary type of infection and from an exogenous source. In a study of scarring at the apex, Baldwin (2), Petroff (3), and Gardner (4), of the Trudeau Foundation, are of the opinion that the infection reaches the apex through the blood stream by way of the pulmonary

artery. They subscribe to the hypothesis that the hilus node, in the active stages of the primary infection, is unable to hold all the tuberculosis bacilli that lodge there and consequently that the bacilli are thrown off into the thoracic duct and thus into the pulmonary artery. They do not attempt to explain why the infection takes place only at the apex.

As this study progressed, it was seen that there was a definite correlation between the finding of calcification in the hilus nodes and apical scarring. Because of this correlation it was thought that, in conjunction with the study of apical scarring, a similar type of study of calcification of the primary infection in hilus nodes would be of value. Consequently both the excised lungs and the roentgenograms of the lungs taken prior to death were studied from the standpoint of the primary infection.

The incidence of primary infection has previously been studied by various investigators. However, the majority of these studies have been carried on among selected groups in various parts of the country. At the Mayo Clinic a fairly uniform cross-section of the rural and metropolitan population of the country is seen. Possibly a comparison of this study with a similar study made some years previously could be carried out to determine roughly the comparative incidence of primary infection.

The calcified, or caseous, hilus node is generally considered to be the most common evidence of primary tuberculosis. However, calcification of a primary focus must be distinguished from broncholiths, phleboliths, and calcific deposits in the bronchial, tracheal, or costal cartilages, or in old pyogenic inflammatory foci. Probably the most common opacity in the roentgenogram mistaken for calcification of the primary focus is that formed by the shadow of the cross-section of a blood vessel. Generally these are all readily distinguished from calcification of the primary focus.

Robertson (16), in a study of the incidence of tuberculosis in patients dying from causes other than tuberculosis, found evidence of it in 1,000 cases at necropsy, or 79.9 per cent. Opie and Andersen (17), in 1920, with the aid of roentgenologic examination, found in a large series of cases evidence of tuberculosis in practically 100 per cent of excised lungs examined in necropsy specimens more than 20 years of age.

Exact figures concerning the incidence of primary infection as found by roentgenograms of the chest are exceedingly hard to obtain. Farrell (18) found an incidence of 77 per cent in patients more than 14 years of age. Ickert (19), using a fluoroscope, found calcification of the primary lesion in 58 per cent of patients more than 14 years of age, and in 20 per cent of school children. Opie (20) has stated that approximately 20 per cent of normal adults will show calcification of the first lesion of tuberculosis in one set of anteroposterior stereoscopic roentgenograms.

Occasionally one will find an adult type of tuberculosis of the lungs with no evidence of a primary focus. It has been shown a number of times that the primary focus can heal without leaving any evidence of its having been there, or that the evidence may be so small as to escape observation.

From the clinical standpoint, probably the most important calcification in the thorax is in the child and the young adult. Robertson (21) has shown that tuberculous lesions in persons less than 30 years of age are found to be active three times as often as in persons more than 30 years of age. In the roentgenograms of 1,164 contact children, J. A. Meyers (22) was able to find calcification in 947. All of these children had been exposed to active tuberculosis. This is an extremely high percentage, especially for children, and shows the significance of calcification of the primary focus in children and young adults. From numerous studies of this sort it can be readily seen that calcification of the primary focus in young persons should be looked on with concern and an effort made to locate the source of the infection.

MATERIALS AND METHODS USED

The materials used in this study were taken from the Department of Pathologic Anatomy and the Department of Roentgenology of the Mayo Clinic. In order to obtain a large enough series of cases for this study the protocols of 350 necropsies performed in the last six months of 1936 and the first three months of 1937 were used, together with 330 additional necropsies personally observed by one of us (E. A. A.) during the period of the six months from April 1 to Sept. 30, 1937, inclusive.

At necropsy the excised lungs were first examined at the apexes for evidence of apical scarring. This could be determined by sight or by palpation with the tips of the fingers. The scar, if found, was then sectioned and the thickness of the lesion noted, and also the appearance of the parenchyma of the lung beneath. In the majority of cases a section was put in fixing solution to be stained with hematoxylin and eosin, and was later studied under the microscope. Next the hilar regions of both lungs in which no evidence of calcification in the nodes could be found were taken to the Section on Roentgenology and roentgenograms were made to determine if there was calcification that had been overlooked at the initial examination of the excised lung. In all cases, whether or not evidence of tuberculosis could be found in the hilus nodes, the spleen, liver, and mesenteric nodes were examined for healed tuberculosis.

Following necropsy the roentgenograms, if any had been made at the time of the patient's clinical examination, were reviewed for evidence of healed primary infection and of apical scarring. If there was no evidence at necropsy of healed primary infection or no evidence of calcification of the primary focus in roentgenograms of the chest, the plates of the pelvis or abdomen, if any had been taken, were reviewed to determine if there was calcification in the mesenteric nodes that had been overlooked at necropsy. All the



Fig. 5.



Fig. 6.

Fig. 5. Roentgenogram of chest showing irregular opacity at extreme apex which, at necropsy, proved to be an apical scar.

Fig. 6. Roentgenogram of chest showing calcification in an apical scarring which was proved at necropsy.

roentgenograms of patients whose necropsy records were obtained from the protocols were also reviewed to determine the presence or absence of apical scarring and healed primary tuberculosis of the lungs.

All the sections of apical scars taken at necropsy were examined microscopically to determine the activity of the lesion and the extent of the scarring. Many of the nodes in which tuberculosis could not be ruled out macroscopically were sectioned and examined microscopically.

RESULTS

The results of study of the excised lungs personally done coincide very closely with the statistical data obtained from the protocols; therefore, the statistical data in the tables and graph herewith include both studies.

Table I shows the number of necropsies in the various age groups and the percentage of cases of calcification of the hilus node in these groups. Out of this group, 582 roentgenograms of the chest were obtainable. In the 582 roentgenograms, calcification of the primary infection was found in 112, or 19.2 per cent. This includes either the hilus node or the Ghon lesion or both. In the 582, there were only 54, or 9.3 per cent, that had visible calcification of the hilus nodes in a single set of anteroposterior stereograms.

Table I also shows the incidence of apical scarring in the various age groups, the scarring being graded from 1 to 4. Grade 1 is minimal but shows definite scarring both macroscopically and microscopically, and Grade 4 is scarring which covers the entire cap of the apex and approaches 1 cm. in thickness. Among the 582 roentgenograms, there was definite scarring in 92, or 15.8 per cent.

Table II demonstrates the incidence of scarring when no calcification of the hilus node could be found. In 12 instances it was possible to make a roentgenogram of the region of the hilus, and in no instance was calcification of the hilus node found. In two instances calcification of the mesenteric nodes was found both in roentgenograms and at necropsy: in neither instance was calcification of the hilus node found. In only 62, or 9.1 per cent, of the 680 pairs of lungs excised was there no scarring when there was calcification of the hilus node.

In Figure 7 the percentages by age groups of calcification in the hilus node and of apical scarring are charted together to show how closely they follow each other.

There were 25 cases in which a calcified tubercle of both the spleen and the liver was found. In all except two of these cases there was apical scarring, and in both the latter there was calcified tubercule of the spleen alone. In all there were calcified nodes and in only one was there apical scarring. In three cases tuberculosis of the liver was found alone; in all three there were apical scarring and calcified hilus nodes.

In 12 cases there was an active adult type of pulmonary tuberculosis. In all except two there were scars of Grade 2 or greater, and in all except one there were calcified nodes. Of all the sections studied microscopically, only one showed evidence of activity in the apical scar (Fig. 6).

COMMENT

The inadequacy of the roentgenogram to portray the evidence of primary healed tuberculosis in the chest has been brought out in this study. It is far inferior to the skin test and, of course, to the examination of the excised lung at necropsy.

From Table I it is seen that the incidence of tuberculosis is lower in the younger than in the older age groups. This means either that a greater immunity to the tuberculosis bacillus is developing in the younger age groups, or that they are not coming into contact with tuberculosis bacilli as often. If the incidence of tuberculosis is compared with a similar study carried on in this department in 1923 and 1924, it is quite evident that there is a definite decrease. The two studies were carried out in practically the same manner. There is a decrease in incidence from 79.9 per cent to 64 per cent in this 13-year period. At the time of the study in 1923 and 1924 the

Ghon complex was the sole evidence of primary tuberculosis looked for and apical scarring was not considered.

The most frequent site of healed primary tuberculosis found at necropsy is commonly stated to be the calcified or calcareous hilus node. However, the healed lesion at the apex of the lung was the most common lesion found in this series of excised lungs. Among possible reasons for this contradiction is that the scarring at the apex is a lesion which can be seen and felt readily, but the calcified hilus node is often difficult to find. Calcification in the hilus node is frequently pin-head in size and could easily have been overlooked, even with the aid of a roentgenogram of the hilar region. It is probably a scientific impossibility to find all the evidence of healed primary tuberculosis in a lung. If it had been possible to make roentgenograms of each hilus region in the cases in which no calcification was found, the frequency of healed hilar tuberculosis would probably have approached that of apical scarring.

As has been pointed out by various investigators, the presence of a Ghon complex in the child and the young adult should be looked on with a certain amount of apprehension. This was brought out in two cases in this series in which two young persons who died of tuberculous meningitis had calcareous hilus nodes and Ghon tubercles, both noted by roentgenogram. The finding of calcification in the roentgenogram in each case of this type should be an added aid in the diagnosis. Many more of the calcifications in the hilus nodes could be seen on the roentgenogram if oblique views were also taken.

From Figure 7 it can be seen that calcification of the hilus node and apical scarring have very much the same type of curve. Apical scarring occurs five times more often with hilus node calcification than it occurs without it. From this it can be seen that there possibly might be a direct connection between these two manifestations of tuberculosis.

The scarring has no apparent direct

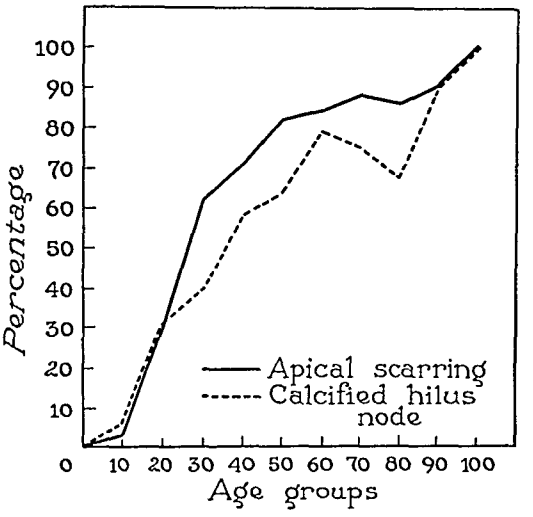


Fig. 7. Percentages by age groups of apical scarring and calcification of the hilus nodes. The same general curve is seen for both.

connection with the Ghon tubercle, for the latter is in the middle or lower lobes in more than 50 per cent of the cases. In two cases found in this study the Ghon tubercle was 2 to 3 cm. from the apex, yet there was no evidence of apical scarring.

Opie and Andersen (17) expressed the opinion that apical scarring is a secondary exogenous infection, mainly because they found it active more often than the hilus node and also because it occurred later in life than the primary infection with its calcified hilus node. This study has brought out two counter-arguments to this conclusion, namely, the apical scarring has been found to be notably benign, and the apical scarring has been found just as often as calcification of hilus nodes in the earlier age groups, if not more often. The only explanation for these discordant findings is the fact that Opie and Andersen's (17) investigations were carried out 17 years ago and in a metropolitan area.

Apparently there is also a correlation of healed miliary tubercles of the spleen and liver with the calcified hilus node and apical scarring. In 25 cases in which healed miliary tubercles of the liver and spleen were found, only two did not have apical scarring, and only one did not have a calcified hilar node. In all seven cases in which tubercles of the spleen were found

alone, there were healed hilus nodes, and all except one had apical scarring. Apical scarring and calcification of the hilus node were found in all five cases in which healed tubercles were found in the liver and not in the spleen. It is interesting to note in this connection that the apical scarring in these cases was more extensive than that commonly found.

To prove scientifically the route of tuberculosis bacilli in gaining access to the apex would be a difficult task and is beyond the scope of this paper. However, a number of facts brought out in this study seem to imply that the infection is lymph-borne. Some investigators have expressed the opinion that the infection is brought about by retrograde lymph flow, due to obstruction of the lymph channels caused by the primary infection. No scientific demonstration of this type of infection has ever been made under the conditions of velocity and pressure that exist in the living body. To find foci of infection peripheral to the node is by no means evidence that the infection occurred by retrograde course. If this did take place, it would seem that it would have to do so by successive implantation of foci and thus, by continuity, run backward against the lymph flow. However, in this type of spread these foci are not found along the lymphatic channels, as Baldwin (2) has shown.

There is a possibility that apical scarring may occur from an exogenous source by an air-borne route, as Opie (1) has suggested. However, a number of points brought out in this study contradict this hypothesis. There is little doubt that apical scarring is a secondary infection and thus the tuberculosis bacillus is implanted in an allergic lung. The pathologic picture is one of a healed diffuse exudative process. It is not the well circumscribed lesion such as is seen in the healed primary parenchymal lesion of tuberculosis. It has been shown a number of times that it takes comparatively large amounts of tuberculosis bacilli to set up an infection in such a lung, and it does not seem possible that 76 per cent of the population would inhale enough bacilli to cause such an infection. The majority of air-borne infections in allergic lungs are first seen in the infraclavicular regions in the outer quadrant of the lung-field and not in the extreme apex.

The possibility that tuberculosis bacilli are brought to the apex by way of the blood stream should be considered. This would adequately explain many of the observations in this study as well as those of other investigators who have become interested in apical scarring. In the past little attention has been paid to the thoracic duct as a medium for the spread of infection. Washburn (23) has recently shown that spread of infection by this channel really does occur. It seems highly probable that a lymph node in the hilus of the lung during the active stage of the tuberculous infection could become so engorged with tuberculosis bacilli that it would be unable to hold them, and consequently some of them would be passed over into the thoracic duct. It has been shown that

TABLE I.—INCIDENCE OF CALCIFICATION IN HILUS NODE ON GHON TUBERCLE AND OF APICAL SCARRING BY AGE

Age Groups	Number of Necropsies	Percentage of Calcification	Instances of Special Scarring Grade 1	2	3	4	Total	Percentage
1– 9	33	6.0	1	0	0	0	1	3.0
10–19	26	30.7	7	1	0	0	8	30.8
20–29	50	40.0	25	5	1	0	31	62.0
30–39	67	58.2	33	10	5	0	48	71.6
40–49	120	62.5	63	32	2	2	99	82.5
50–59	147	79.5	67	50	6	1	124	84.4
60–69	160	75.0	65	65	10	1	141	88.1
70–79	66	79.6	28	22	7	0	57	86.4
80–89	10	90.0	3	3	2	1	9	90.0
90–99	1	100.0	0	0	1	0	1	100.0
Total	680	64.0	292	188	34	5	519	76.3

even in the calcified hilus node, active tuberculosis bacilli can be found in 20 per cent.

Sweany (24), in a study of tuberculous lymph nodes in all stages of tuberculosis, points out that it would be easy for the bacilli to get into the pulmonary circulation. Once the tuberculosis bacilli are in the thoracic duct they are carried directly into the pulmonary artery by way of the subclavian vein and throughout the entire lung-field.

Tuberculosis of the joints, kidneys, liver, and spleen is commonly thought to be brought about by bacilli being carried there in the blood stream, and it is probably no more illogical to postulate that the tubercle bacilli are carried to the apex in this manner. Why the bacilli do not set up miliary tuberculosis when the bacillemia occurs has never been explained. The selective localization of the tuberculous infection at the apex has long been a subject of scientific investigation. No one of the various investigations has established, to the satisfaction of all, the reason why tuberculosis selects the pulmonary apex.

TABLE II.—INCIDENCE OF APICAL SCARRING WITHOUT CALCIFICATION OF HILUS NODES

Grade	Number	Percentage of Instances of Apical Scarring of this Grade
1	87	29.8
2	30	16.0
3	4	11.8
4	0	0
Total	121	23.3

The possibility of poor lymphatic drainage at the apex has also to be considered in this connection. Miller (25), in a study of the lymphatics of the lung, has shown that the periphery of the lung parenchyma drains toward the pleura and not toward the hilus. In this study the apical scarring was never found on the visceral pleura where the first rib compressed the pleura and the immediate lung parenchyma. Also, it is known that tuberculosis may infect any part of the lung that has its lymphatics blocked by general silicosis.

The correlation of tuberculosis of the



Fig. 8.



Fig. 9.

Fig. 8. Roentgenogram of chest showing the shadow of the subclavian artery.
Fig. 9. Roentgenogram of the chest showing the shadow of Sibson's fascia.

tonsils and cervical nodes with apical scarring is probably better explained by the infection following the lymphatic channels that drain into the right lymphatic duct and thoracic duct than by the manner in which Van Zwaluwenburg and Grabfield (6) and Grober (8) believed it reached the apex. It is not probable that two layers of adjoining pleura could be infected and still so few cases occur with adhesions between the two surfaces.

If the explanation of apical scarring as a result of infection through the blood stream is accepted, then apical scarring could be brought about by almost any focus of tuberculosis in the body. There were 121 cases in which it was not possible to find calcification in the hilus node in conjunction with the apical scar. Possibly the source of the apical lesion in some cases was in the tonsils, the cervical nodes, or the mesenteric nodes.

It has been shown by experimental study in animals that tuberculosis bacilli are carried into lymph channels and the vascular system by macrophages. It is then possible for tuberculosis bacilli to gain access to the arterial circulation and thus set up minute infections in the liver and spleen. Why the bacilli localize in the spleen and liver in the majority of cases can be explained only on the basis of selective localization.

From the roentgenologic standpoint, the apical lesion is overlooked in the majority of cases. The portrayal of apical scarring in the roentgenogram is definitely a different shadow than that of an old healed fibrotic parenchymal lesion and should not be confounded with such an opacity. Seldom is calcification seen in an apical scar.

However, there are two structures at the apex that might be confounded with apical scarring. The first is the shadow of the subclavian artery (Fig. 8). This shadow is a homogeneous opacity with a sharply defined lower border, concave downward. It is seen beneath the third rib posteriorly on the left side; however, it may also be seen on the right side. If the roentgenogram is made with the tube centered over the mid-chest, the shadow of the artery may be seen closer to the second rib posteriorly. A vertical continuation passing to the aortic arch medially is often seen.

The other confusing structure is Sibson's fascia (Fig. 9). This fascia extends from the transverse process of the seventh cervical vertebra on the same side to the inner margin of the first rib. It is seen only within the arc of the first rib and in the posterolateral quadrant of the apical cupola. The direction is forward and outward, and rather more anteroposterior than lateral. The visceral margin is sharply defined and is usually slightly concave toward the lung-field. The density may be so light as to be hardly visible. The shadow can never be seen to cross the first rib, and on stereoscopic examination it is distinctly not a bony structure.

SUMMARY

An infrequently studied tuberculous lesion of the pulmonary apexes has been described. Its possible connection with other healed tuberculosis in the body is also considered. Apical scarring, as reviewed in the roentgenogram, must be distinguished from normal apical structures. There appears to be a general decline in the incidence of primary tuberculous infection in the chest.

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OSTEOPETROSIS: A CASE REPORT[1]

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OSTEOPETROSIS (syn. marble bones, *Marmorknochenkrankheit,* osteosclerosis fragilis generalisata, Albers-Schönberg disease) is a disease primarily characterized by increased density and thickness of the cortical and spongy portions of the bones with encroachment on the medullary cavity. It involves the entire skeleton. Roentgenologically it is distinguished by increased opacity of the bones which appear heavy and lacking in structural detail. The posterior clinoids are usually thickened and clubbed, the skull bones show increased density, and there are frequently peculiar alterations in the contours of the long bones. There is often a concomitant anemia of the myelopthisic type with enlargement and myeloid changes of the spleen, liver, and lymph nodes.

Multiple fractures are frequently found; occasionally osteomyelitis, optic atrophy, hydrocephalus, and subarachnoid hemorrhage are other more or less frequent manifestations of the disease.

The condition was first described by Henck in 1879, and first demonstrated roentgenologically by Albers-Schönberg in 1904. In 1934, McCune and Bradley (5) presented a review of the literature up to 1933 and reported a case. In their review they cited 68 cases, including their own case and two reported after their paper was accepted for publication.

Since 1933 we have been able to find reports of seven other cases (2, 3, 6, 7, 8, 9, 10).

McCune and Bradley stated that in eight cases there were complete, and in four, incomplete, autopsies. In their case rather comprehensive clinical studies were possible but autopsy was denied.

The following case is presented to add another to those recorded, and to present the autopsy report. Unfortunately the postmortem examination was necessarily incomplete in regard to the osseous system.

The male child, J. P. H., was brought to the Wisconsin General Hospital in a moribund condition. The case history was summarized by Dr. K. B. McDonough, of the Department of Pediatrics, and the pathologic findings follow.

The patient, one year of age, was admitted to the pediatric service of the Wisconsin General Hospital on April 18, 1936. The history, obtained from the mother, stated that she and the child's father were 40 and 39 years of age, respectively, and in good health. There were four siblings, in age 15 to three years, all in good health. There was no history of syphilis, tuberculosis, or blood disease. The infant was born at full term; delivery was spontaneous. The child was very blue at birth and held its head in a "funny" position. He cried with short staccato cries, which did not sound normal, whenever anyone tried to straighten up his head, which was held well back. The infant became jaundiced shortly after birth. This jaundice was present off and on throughout the first year. The infant weighed seven and one-quarter pounds at birth. He had always been under the doctor's care, and seemed to have fever and chills at various times for no apparent reason. He had had convulsions at intervals and these had increased in frequency and severity during the past two months. He was breast-fed for three months and vomited a great deal but the vomiting was not projectile in type. He received a simple milk mixture after the age of three months and was started fairly early on orange juice and cod liver oil. He had also received some vegetable soup and oat meal gruel. These were started late in the first year because the

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infant was unable to eat well. He was unable to sit up and had difficulty in holding his head up. He did not grow as the other children had during the first year of life. The infant had always been constipated and appeared to be unusually irritable. He had a tendency to hold his arms at a peculiar angle behind his head. The mother thought that the baby experienced some pain when his arms were extended or an attempt was made to put him in a sitting position. It was thought that the infant had grown paler during the past two or three months.



Fig. 1-*A*.

Physical examination showed a very pale, markedly undernourished infant who appeared small for his age. His head appeared large in proportion to the rest of his body. He had a definite Mongoloid facies. The eyes were widely spaced and there was a slight exophthalmos. The facies was not that of a hydrocephalic infant. There was a yellowish pallor of the skin. His length was 25 inches. The head was rather large, measuring 47 cm. The anterior fontanelle was open and measured 4 × 5 cm.: the remainder of the fontanelles were closed, as were also the suture lines. The skull felt thick to percussion and a flat note was elicited throughout. The left occipito-parietal region was prominent. Examination of the eyes showed the extra-ocular movements to be normal. Vision was apparently normal. The fundi could not be seen. There were no teeth, although the upper gums were swollen. Excessive lymphoid tissue was present in the pharynx. The infant breathed through his mouth. The ears were normal. The neck was held rather rigidly and when the infant was raised to a sitting position he held his head erect and cried loudly. The

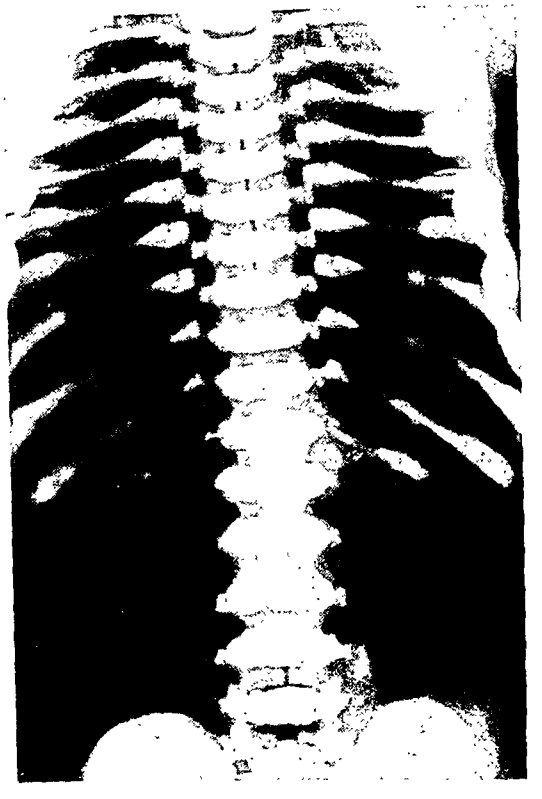


Fig. 1-*B*.

anterior and posterior cervical lymph nodes were enlarged. The thyroid was not palpable. The chest was symmetrical but expansion seemed to be limited. The costal margins were flaring. The heart and lungs were normal to physical examination. The abdomen was prominent. The spleen was firm and smooth, and palpable to the crest of the ilium. The liver edge was palpable 5 cm. below the costal margin. There was a generalized adenopathy. Examination of the extremities showed no limitation of movement and the infant was able to kick and grasp objects without difficulty. The wrists and ankles were not enlarged. Neurologic examination showed all the reflexes to be hyperactive. During the examination the infant had a convulsion, lasting about one minute, which was characterized by opisthotonos and tetanic spasm of all the extremities. Before any laboratory studies or x-rays could be obtained, the infant experienced a very severe convulsion. He became very cyanotic and before measures could be instituted to stimulate respirations, the infant expired.

AUTOPSY

The body was that of an emaciated male infant 64 cm. in length. Enlargement of the cervical, axillary, and inguinal lymph nodes was noted, and these nodes were soft. The abdomen was distended. The spleen was considerably enlarged and weighed 210 gms. The cut surface was dark red and firm. The pulp was apparently increased in amount, and the malpighian corpuscles were not visible. The liver was somewhat enlarged, weighing 730 gms. The cut surface was pale with a slight yellow tint. The mesenteric lymph nodes were enlarged and soft.

The bone of the skull was 8 mm. thick, dark red and mottled in appearance. There was organizing hemorrhage between the skull and the dura, and these had to be



Fig. 2-A

Fig. 2-B.

separated by blunt dissection before the calvarium could be removed. Except for a well marked edema of the leptomeninges over the frontal and parietal lobes, the brain showed no pathologic change.

The bones of the whole body were increased in density. The cortex was thickened and the marrow cavities were almost completely filled by a network of heavy bony trabeculæ.

Essential Microscopic Findings.—The lymph nodes, spleen, and liver were the sites of extra-medullar blood formation, clusters of primitive cells being found in all these organs. Eosinophilic myelocytes were especially prominent.

The femur could be sectioned only after long decalcification. The cortex was thick and the medullary cavity was almost completely filled by heavy, bony trabeculæ. In some fields there were irregular areas of newly formed bone, staining blue with hematoxylin and eosin. The bone marrow was scanty, and in some places was replaced by fibrous tissue. Cellular detail was not well preserved on account of the long decalcification necessary, but here and there one could recognize an osteoclast. Groups of osteoblasts were also present.

Anatomical Diagnosis.—Idiopathic osteosclerosis (Albers-Schönberg's disease), with compensatory myeloid change in the lymph nodes, spleen, and liver.

COMMENT

Several roentgenograms were obtained before and after the postmortem examination (Figs. 1–3). The roentgenograms exhibit findings similar to those of the case of McCune and Bradley (5). These authors should be consulted for a complete discussion of the findings. There is in this case the additional roentgenographic finding of markedly increased thickness of the right parietal region with localized tumefaction (Fig. 3-*B*), general increase in the thickness of the skull, and increased density and lamellation of the periosteal or pericortical new bone.

The lack of similarity of the roentgenographic picture in the infant and in the older child and adult is probably associated with alterations in the activity of bone formation incident to growth. It is evident in this case, also, that the changes began *in utero*, and that at or about the time of birth some alteration in the pathologic process occurred. The presence of scurvy need not be hypothecated, and rickets is unlikely. A parallel striation of the metaphyseal portions of the long bones is very distinct. Not stressed previously are the perpendicular striations of the "rims" of the ilia and the texture of the skull. Clubbing of the clinoids is particularly evident in the anterior processes. On the original films of the skull a thin line of periosteal



Fig. 3-*A*. Fig. 3-*B*.

new bone formation similar to that about the shafts of the long bones is evident.

The disposition of the density in the vertebral column is interesting.

The recently published reports (1–10) on the association of fluorine intoxication and osteopetrosis in the adult suggest roentgenologic investigation of infants and children in regions where fluorine ingestion is known to be considerable. The data in this case are insufficient to determine the possibility of fluorine as an etiologic factor.

The possibility that cases such as the one presented and of infants with similar findings represent an entirely different disease of distinct pathogenesis from the adult types must not be disregarded.

SUMMARY

1. A previously unreported case of osteopetrosis in an infant is presented, with the pathologic findings and a series of illustrative roentgenograms.

2. There is some question as to whether all cases reported as osteopetrosis fit into a common clinico-pathologic syndrome.

3. Investigation of infants and children in regions where some degree of fluorine ingestion is known to occur is suggested as one means of determining the etiology of this condition.

4. Periosteal involvement is, contrary to statements in the literature, present in this case.

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BENIGN DUODENO-COLIC FISTULA

WITH REPORT OF TWO CASES[1]

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DUODENO-COLIC fistula is a rare finding and of the few cases reported in the literature only two have been described as being due to benign lesions; it is an abstract of these two cases which will be given in this paper. The other cases were all due to a carcinoma of the transverse colon perforating into the duodenum. Other cases may have been reported in connection with other lesions of the gastro-intestinal tract, but I have been able to find only two cases reported under the heading of "duodeno-colic fistula" from 1885 up to the present time.

The etiology of benign duodeno-colic fistula is usually a perforation of a duodenal ulcer into the transverse colon with a resulting fistula between the two. Other benign lesions may cause a fistula between the duodenum and colon, but none could be found reported in the literature. The absence of a typical history of duodenal ulcer does not exclude this as the cause, for we are all familiar with cases of duodenal ulcer in which the only sign or symptom of the ulcer may be a sudden hemorrhage, with subsequent roentgenography demonstrating the presence of an ulcer though the patient still presents no real symptom of the ulcer except for the hemorrhage.

The fistula may involve any part of the duodenum because of the close relationship between the duodenum and the transverse colon.

Ulcers of the second and third parts of the duodenum are probably more common than is generally realized. Hauser (1), using the combined statistics of five authors on the location of *perforated* duodenal ulcers, lists 274 as being located in the first part, 12 in the second part, and five in the third part. This indicates that almost 6 per cent of perforated duodenal ulcers are located in the second or third part of the duodenum. These figures are probably high for a general average, as there may be a greater tendency for ulcers of the second and third parts of the duodenum to perforate than for ulcers of the first part. These figures, however, do indicate that ulcers of the second and third parts of the duodenum are more common than we realize. Accurate statistics on the frequency of ulcers beyond the first part of the duodenum are not available, because ulcers here are more difficult to detect roentgenologically than in the first part and they are usually not operated upon unless they perforate.

The patient with a duodeno-colic fistula may or may not give a history of a sudden abdominal pain. A persistent diarrhea with rapid loss of weight, in spite of a voracious appetite, is quite typical of a duodeno-colic fistula. This history is common for any fistula between the colon and the upper gastro-intestinal tract and is not specific for a duodeno-colic fistula.

Blondeau and his co-workers (2) reported a case of a 55-year-old man who came to them for an incorrigible diarrhea, the yellowish liquid containing undigested alimentary débris. His symptoms were not very pronounced, there being merely a feeling of some epigastric heaviness after meals. He had sometimes, but not always, colicky pains but without much suffering, and without the insistence of his family would not have been roentgenographed. On examination there was noted a pronounced emaciation but with no evidence of an intra-abdominal tumor. The liver was hard and slightly enlarged. A barium meal showed rapid emptying of the stomach. The first few mouthfuls of the meal

[1] Presented before the Twenty-fourth Annual Meeting of the Radiological Society of North America, at Pittsburgh, Nov. 28–Dec. 2, 1938.

entered the duodenum rapidly and cascaded into the transverse colon. About 90 per cent of the meal passed into the transverse colon, with the remainder passing through the duodenum into the jejunum. In one hour the stomach was empty. There was definite tenderness to palpation at the sub-hepatic angle. Operation was advised but was refused by the patient. During the following two months the diarrhea became less severe and the patient improved generally. The following month he presented signs of pyloric obstruction, confirmed by roentgenoscopy. He continued to refuse operation and died one week after the last examination.

This case, although it was not proved by operation, is included as it had been reported as a benign fistula. The history of the patient during the period of his observation and with the occurrence of pyloric obstruction are much against a benign fistula, and I believe this case was probably due to a cancer of the colon perforating into the duodenum.

Rees (3) reported a case of a woman 62 years of age, whose past history was negative except for typhoid fever 28 years previously. She complained of indigestion and a sense of weakness and soreness in the pit of the stomach, which had been present for eight or nine months. About two hours after meals she became distressed and complained of gas and occasional vomiting. Laxatives gave her temporary relief. She had lost 15 pounds in weight. A Graham-Cole test revealed no filling of the gall bladder. Roentgen examination with a barium meal revealed no evidence of pathology of the esophagus, stomach, or duodenum. The sphincter of Oddi was incompetent and permitted the barium to pass into the hepatic ducts. The colon was not examined with a barium enema. Exploratory celiotomy revealed slight thickening of the gall bladder but without



Fig. 1.



Fig. 2.

Fig. 1. Case 1. A barium enema showing filling of colon, duodenum, stomach, and jejunum. Arrows indicate filling defect in the colon at the site of the fistula.

Fig. 2. Case 1. Roentgenogram made after evacuation of the barium enema. Note barium remaining in the stomach, duodenum, and jejunum. Arrows point to deformity of first part of duodenum by active ulcers.

gallstones. The common duct was dilated to about twice the normal size. A dense band of tissue was found extending anteriorly from the middle of the lateral aspect of the descending duodenum and the proximal portion of the horizontal duodenum to the upper border of the transverse colon. Further dissection revealed a fistulous tract, 5–6 cm. in length, extending from the transverse colon to the duodenum just distal to the ampulla of Vater. Microscopic examination of the excised fistulous tract showed the upper half of the tract to be lined with epithelium similar to that in the duodenum, while the lower half of the tract was lined with epithelium similar to that in the colon. Rees assumed that the fistula must have resulted from a perforation of an ulcer in the colon during the attack of what was thought to have been typhoid fever 28 years previously.

This case was not diagnosed by roentgen examination of the stomach and duodenum. If a barium enema had been given, it is thought that there would have been no difficulty in demonstrating the fistulous tract. This point, which has been mentioned previously in the literature on many occasions, should again be emphasized. Any fistulous tract involving the colon can be most accurately and easily detected if a barium enema is given. If the examination is limited to the barium meal, the fistulous tract may not be demonstrated. Although many fistulæ between the colon and the stomach or small intestine have been demonstrated with a barium meal, a failure to demonstrate the fistula with the barium meal does not exclude the possibility of its being present.

Of the above two cases abstracted from the literature, one was diagnosed on roentgen examination but was not confirmed by operation or postmortem examination, and the other one was not diagnosed on roentgen examination but was found at operation. The following two case reports are, as far as I have been able to find, the only two such cases which have been diagnosed by roentgenography and confirmed by operation. In the second case the roentgenologic and surgical diagnoses were confirmed by postmortem examination.

The two cases reported in the literature and also my two cases come within the "carcinoma age," and the probability of cancer must be differentiated from a benign lesion. The roentgen examination can strongly suggest the benign nature of the lesion, but a cancer cannot be definitely excluded except by operation. The accurate differentiation between a malignant and a benign fistula by roentgen examination is not essential, because surgery is the only method of treatment in either case and is necessary to establish the correct diagnosis.

After operative resection of the fistulous tract, the diarrhea disappears, the patient gains weight rapidly, and soon returns to normal health. The cure of these patients by surgery is quite dramatic.

REPORT OF CASES

Case 1. F. H., a 58-year-old Catholic priest, was referred to the Leonard Morse Hospital, Natick, Mass., on Nov. 2, 1931, for roentgen examination of the gastrointestinal tract. His chief complaints were loose, watery bowel movements and loss of weight.

When about twenty years of age the patient had a bilateral excision of the glands in his neck. The past history is otherwise unimportant.

Three years before entry (that is, in 1928), he began to have loose, watery bowel movements, usually occurring twice a day, with short intermissions up to the present time. There is no history of blood in the stools. Seven months before examination he began to lose weight and there was an onset of vomiting, usually waking him up in the night and occurring several times during the week. These attacks followed the eating of greasy or fried foods or on a day when he had had no bowel movement. The vomitus consisted chiefly of watery material but occasionally there were particles of food eaten at breakfast time, perhaps sixteen hours previously.

No blood was seen in the vomitus. He had lost about twenty-five pounds in weight during the seven months before his entry into the hospital.

Physical examination showed an extremely emaciated elderly man with a slightly injected pharynx, normal heart, lungs, and abdomen. The blood pressure was 85/50; the white blood count 5,300 per c.c.; the red blood count 2,500,000 per c.c., and the urine negative but with a renal function of 40 per cent.



Fig. 3. Case 2. A barium enema showing filling defect in the colon at the site of the fistula to which the arrows point. (*C*) colon; (*D*) duodenal cap.

Roentgen examination with a barium enema showed no evidence of pathology in the descending colon. In the transverse colon, just distal to the hepatic flexure, there was a filling defect and at this point the barium was seen to enter the duodenum at the juncture of the first and second parts and thence into the stomach with only a small amount of the barium passing into the colon proximal to this point (Fig. 1). The filling defect in the colon was at the point of the fistulous opening and it did not have the characteristic ragged outline usually seen in a carcinoma of this type. The patient was then allowed to expel the enema, after which, examination revealed a large amount of barium remaining in the stomach and duodenum (Fig. 2). Some of the barium had passed from the duodenum into the jejunum. A barium meal was then given but I could not see the barium pass from the duodenum into the colon through the fistula. The first part of the duodenum was deformed by an ulcer. The fistulous tract entered the duodenum just distal to the deformity caused by the ulcer. A diagnosis of a duodenal ulcer and a fistula between the duodenum and colon was made. The cause of the fistula was thought to have been a perforation of a duodenal ulcer into the colon or a perforation of a carcinoma of the colon into the duodenum.

The patient was referred to the Peter Bent Brigham Hospital, in Boston, for operation. A barium enema at that hospital confirmed my findings. The patient was operated upon by Dr. David Cheever and I am indebted to him for his permission to use his operative and follow-up notes, as follows: "A right paramedian incision was made. The liver appeared normal. The gall bladder was buried in adhesions. A hasty examination of the stomach and coils of intestine generally revealed nothing abnormal. Just distal to the pylorus there was marked deformity, with induration of the first part of the duodenum, and subjacent to this could be felt a definite small mass, deeply seated behind the colon. In order adequately to unravel and identify this it was necessary, first, to dissect the gall bladder free from adhesions by sharp dissection, mobilizing the second part of the duodenum from the posterior parietes and mobilizing the hepatic flexure of the colon, and to free the ascending colon from its connections. After this it could be demonstrated that the hepatic flexure of the colon was firmly attached to the first part of the duodenum by a tube-like structure about the size and thickness of a small pencil and about one centimeter

in length. After complete dissection it was possible to pass the finger completely around the fistulous tract. The adjacent colon felt normal. The first part of the duodenum, proximal to the fistula, was deformed and there were apparent traces of new ulcerations just distal to the pylorus, as well as the one which presumably caused the fistula by perforation, which was more on the inferior aspect of the first portion. The fistulous tract was ligated and divided with the cautery. A posterior gastro-jejunostomy was done in the usual manner."

The patient made an uneventful recovery. At the time of the operation he weighed 100 pounds. He gained nine pounds in weight while he was in the hospital and three weeks after his discharge from the hospital he had gained a total of 16 pounds. By the end of the first year after the operation he had gained over thirty pounds. At the last report, six years after the operation, he was in good health, and examination at that time revealed nothing abnormal. He had lost some weight during the last year but this I believed to be the usual loss which occurs with advancing years.

No microscopic examination was made as no tissue was removed at the operation. The six years of normal health following the operation I think definitely eliminate cancer as the cause of the fistula and the finding of acute ulcerations in the first part of the duodenum, at the operation, indicates also that the fistula must have been due to a perforation of a previous ulcer of the duodenum.

Case 2. R. H., the patient, a 46-year-old male machinist, was referred to my office on June 17, 1937, for roentgen examination of the gastro-intestinal tract by Dr. Thomas R. Donovan. His chief complaints were a severe diarrhea and a marked loss of weight.

The family and past histories were essentially negative.

For the past 15 years he has suffered from a persistent "indigestion," with irregular periods of freedom from symptoms. He has had pain in the epigastrium most of this time, which came on about one to one and one-half hours after meals and was relieved by eating food or by taking baking soda. He has not been under any regular medical treatment during this time. His greatest weight during this period had been 138 pounds. About one year previous to his visit to me he had developed a sudden, acute, agonizing pain in the epigastrium just at the completion of a 150-mile automobile trip. Upon reaching his destination he immediately went to bed, where he was attended by a physician who administered opiates to relieve the pain. Within a few hours the pain was relieved and by the end of the week the man felt much better and his previous digestive symptoms had disappeared. A diarrhea, between six and nine movements a day, began at that time, and he developed a voracious appetite. He ate large quantities of any food that was available, but, in spite of this, he experienced a rapid loss of weight and strength. For the six months previous to the present examination he had been under medical treatment for the persistent diarrhea, but examinations of his stools were always negative. The diarrhea has been so severe as to occasion pain and soreness in the rectum. He states that he has been unable to work for the past three years because of his illness. He has lost 46 pounds in weight.

Physical examination showed a man of middle age, emaciated to a marked degree. His weight was 94 pounds. His heart and lungs were negative. His abdomen was flat and not tender to palpation and there were no palpable masses. There was excoriation about the rectum, with marked painful spasm of the sphincter which prevented a satisfactory rectal examination. The remainder of the physical examination was negative.

The laboratory examinations revealed a normal urine; 6,600 white blood corpuscles per c.c.; 3,600,000 red blood corpuscles per c.c., and the hemoglobin to be 70 per cent (Tallquist).

Roentgen examination of the colon with

a barium enema (Fig. 3) revealed a smooth filling defect in the transverse colon distal to the hepatic flexure. At this point the barium column passed through a fistulous opening into the duodenum at the juncture



Fig. 4. Case 2. Roentgenogram made after evacuation of the enema which was immediately followed by the barium meal. The arrows indicate the site of the fistula. (*C*) colon; (*S*) stomach, and (*D*) duodenal cap.

of the second and third parts. The barium passed up the duodenum and filled the first part and some of it passed through the pylorus into the stomach. Some of the barium also passed through the third part of the duodenum and downward into the jejunum. Some of the barium passed by the fistulous opening and filled the hepatic flexure, ascending colon, and cecum. The colon distal to the fistulous opening was quite smooth in outline and lacked the usual haustral markings, but there was no evidence of an ulcerative colitis. After the patient was allowed to expel the enema, examination revealed a large amount of the barium to have remained in the stomach and jejunum. A barium meal was then given (Fig. 4), but I could not definitely visualize any of the meal as passing through the fistula from the duodenum into the colon. At this time there was excellent visualization of the colon at the site of the fistula (Fig. 5), yet there was no evidence of a ragged outline to the filling defect such as would usually be seen in a case of cancer. The filling defect was quite smooth in outline, was not palpable, and had the

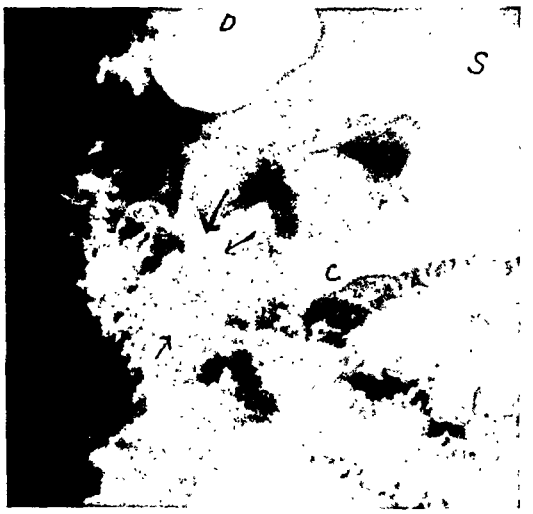


Fig. 5. Same as Figure 4 but showing localized area of fistula. Note the benign appearance of the colon at the site of the fistula.

appearance of a benign lesion. A diagnosis of a duodeno-colic fistula was made and its benign nature was suggested to the patient's physician.

He was operated upon five days after the roentgen examination. Under avertin-ether anesthesia, a high right rectus incision was made. The hepatic flexure of the colon was found to be embedded in a dense mass of adhesions, extending posteriorly and involving the third part of the duodenum and also the distal portion of the second part of the duodenum. The duodenum was quite long and mobile except at the juncture of the second and third portions, where a fistulous tract was found to extend from this part of the duodenum to the transverse colon near the hepatic flexure. The peritoneum was incised along the lateral aspect for a distance of about five centimeters, up to the area of the fistulous tract. After sharp dissection the fistulous tract was seen to be very short and the colon was found to be adherent by its posterior aspect to the upper aspect

of the duodenum. The fistulous tract was divided and the openings into the duodenum and colon were closed. The gall bladder was normal and was filled with easily expressed bile. There were no adhesions about it and, due to the marked emaciation of the patient, the cystic and common ducts were easily identified. The stomach was negative. There was no evidence of an ulcer or other pathology of the duodenum, except for the fistula. The patient had a satisfactory post-operative course for five days and the bowel movements had returned to normal. At this time he complained of pain in the lower abdomen and there was some elevation of his temperature. Rectal examination revealed marked tenderness anterior to the rectum. There was a moderate amount of bulging of the abdominal wall in the right lower quadrant. An incision was made in this area and a large abscess cavity was entered, filled with pus mixed with a brownish foul-smelling fluid. The abscess cavity was in contact with the cecum and extended upward along the ascending colon for a distance of about five centimeters. This cavity extended downward into the pelvis. The cavity was emptied by suction and drains were inserted. The patient did not respond satisfactorily and died twelve days after the second operation. A postmortem examination was performed. The duodenum and colon in the area of the fistula were removed and sent to a pathologist for examination, which revealed several small polyps in the colon near the site of the fistulous opening. No evidence of cancer could be found in the duodenum or colon.

This case gave a typical history of a duodenal ulcer for the past 15 years, with a sudden attack of pain which probably represented the perforation of the ulcer into the colon, as the diarrhea immediately followed this attack. There can be no question but that the fistula was caused by a perforation of a duodenal ulcer, and that the ulcer from which the patient apparently suffered for 15 years was located at the juncture of the second and third parts of the duodenum. The roentgen examination, the operation, and the postmortem examination all failed to demonstrate an ulcer in any other part of the duodenum or the stomach.

SUMMARY

Benign duodeno-colic fistula is a rare finding and only two cases could be found in the literature. One of these cases was not confirmed and, from its history, it was probably not a benign fistula.

The benign fistula is usually due to a perforation of a duodenal ulcer into the transverse colon and it may involve any part of the duodenum.

The diagnosis is not difficult if the colon is examined with a barium enema, but the fistula may not be demonstrated if the examination is limited to a barium meal.

The benign origin of the fistula may be suspected from its appearance on roentgen examination, but operation is necessary to exclude definitely the presence of a cancer.

Two cases are reported here which have been proved by operation and also, in one instance, by postmortem examination, to be due to a perforation of a duodenal ulcer into the transverse colon.

18 Hartwell St.

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DISCUSSION

JOHN D. CAMP, M.D. (Rochester, Minn.): There is very little I can add to this subject because I have never seen such a case and the records at our institution fail to disclose any such lesion on the basis of a perforated duodenal ulcer.

Dr. McPeak has emphasized the fact that it is impossible to determine the benign nature of this fistula prior to surgery and, of course, he also menti

that malignant disease is the most common cause of this condition.

Another not common but not rare cause of a fistula involving the colon is the presence of gall-bladder disease and pericholecystitis with the formation of a fistula between the gall bladder and colon. The duodenum is frequently involved in the inflammatory mass and, in the presence of such a condition, a fistula between the duodenum and colon—and possibly gall bladder as well—may occur.

If the roentgenologist cannot make the diagnosis of a benign fistula prior to surgery, it seems to me that the two cases which Dr. McPeak has mentioned have some very definite clinical evidence which may allow this distinction to be made. In the first place, both patients had a story which was quite typical of a duodenal ulcer, complicated by an acute attack of pain. Such a story, of course, is not common in the presence of malignant disease, and I think this is important in trying to establish the diagnosis, because the surgical treatment of this condition is necessarily a radical procedure, and if we can have some reassurance of its benign nature before operation, radical surgical procedures to effect a cure can be justified.

THE SIGNIFICANCE OF THE ROENTGEN VISIBILITY OF THE HORIZONTAL FISSURE[1]

By PHILLIP T. KNIES, M.D., *Columbus, Ohio*

THE appearance in the roentgenogram of the chest of linear shadows corresponding to the interlobar fissures of the lung is not unusual, and by comparison of such shadows with anatomic specimens a better understanding of the anatomy of the lungs has been attained (10, 5, 24). This has permitted the more accurate localization of pathologic processes seen in the chest roentgenogram in terms of the lobes and fissures which may be involved. Particularly important has been the identification of certain previously puzzling shadows sometimes included among the "unresolved pneumonias," and which have, in some instances, been shown to be due to effusions limited to the pulmonary fissures. The unusual and, at times, bizarre outlines of these shadows are thus easily understood.

By means of its normal position in the horizontal plane, the fissure between the right upper and middle lobes has attracted primary attention in this extension of x-ray critique. Thus, it is obvious that the plane of a fissure which becomes partially radiopaque would more easily be demonstrable when its greatest diameter lay in the axis of the roentgen ray, whereas, if the ray should penetrate it perpendicularly, the result might be only a faint haziness in the area of its projection, difficult of evaluation, if even detectable. In the customary postero-anterior chest roentgenogram, the horizontal fissure is, therefore, in a position, despite its slight superior convexity, to cast its densest shadow, whereas the long or oblique fissures in front of the lower lobes, being intercepted perpendicularly by the x-ray in the greater part of their extent, are not usually noticed on the film unless they contain a considerable amount of fluid, or their walls are grossly thickened by pathologic induration. Such pathology might give rise to the unusual shadows referred to previously, correctly identified through an adequate understanding of pulmonary lobar anatomy, and by the study of the interlobar spaces, in the case in question, by unusual or special technic. At times the lateral extremity of the oblique fissure attains a plane passing anterolaterally from the hilus which permits it to be demonstrated in the usual chest radiogram as an almost vertical line or one passing slightly laterally and cephalad from the diaphragm, approximately one-fourth the distance from the lateral costal margin to the mid-line (Fig. 1). This shadow may be either unilateral or bilateral, and visualization is made possible by the occasional concurrence of the plane of the fissure and the more divergent rays from an x-ray target comparatively close to the film. A lateral projection, however, permits better demonstration of the oblique fissure of the hemithorax nearest the film, and, on the right, its relationship with the horizontal fissure (11). These demonstrations have been presented by several authors (2, 4, 10).

The accumulation of fluid in limited interlobar spaces has been described frequently (1, 6, 9, 13, 17, 18, 20, 21, 22, 23, 25), the usual rotund shadow requiring differentiation from abscess or primary or secondary cancer of the lung. Such accumulations have been accounted for on a hydrostatic basis in cardiac decompensation as a localized form of right pleural effusion, the remaining pleural space having been obliterated by adhesive pleuritis. They have also been considered, at times, to be of inflammatory origin complicating tuberculosis, pneumonia, or rheumatic fever. Practically all such accumulations reported are too well marked to be questionably pathologic, and their problem has been one of differential diagnosis among

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pathologic entities rather than distinction from normalcy.

Although, therefore, it has been recognized that gross shadows of particular location and conformation may represent pathology related to the pleural fissures and the fissural pleuræ, the significance of a much greater number of fine-to-coarse linear shadows in the areas of the fissures has remained in doubt. Particular debate has centered on the pathologic significance of such linear projections of the right horizontal fissure of the lung, which, due to its position, is particularly suited to demonstration without special technic. Some authors (2, 3, 4) have considered these lines to vary in significance only quantitatively from the grosser infiltrations or effusions in the fissures, and to have similar origin, hydrostatic, exudative, or proliferative, as the case might be. Others have considered at least the finer projections to represent the normal pleuræ, which they have thought capable of casting a roentgen shadow when viewed tangentially. Crecelius has been able to show such linear shadows of normal fissural pleuræ in roentgenograms of lungs removed from the chest through careful adjustment of the tangency of the central roentgen ray to the pleural surface (4). Inouye (8), on the other hand, considered the normal pleura incapable of casting a shadow despite tangency of the ray. Double lines occasionally are seen (Fig. 2), but have been considered to have a significance no different from single ones (2), the fissural pleura merely being tangent in two places to the plane of the roentgen rays.

Anatomico-pathologic correlations have, at times, shown pleural induration apparently to be responsible for the antemortem demonstration of such linear shadows, but in numerous instances no such correlation has been present, the pleura appearing quite normal at autopsy (4). In such instances, the linear roentgen images were assumed to be due to normal pleuræ, a view not taking into account a second possible explanation of apparent

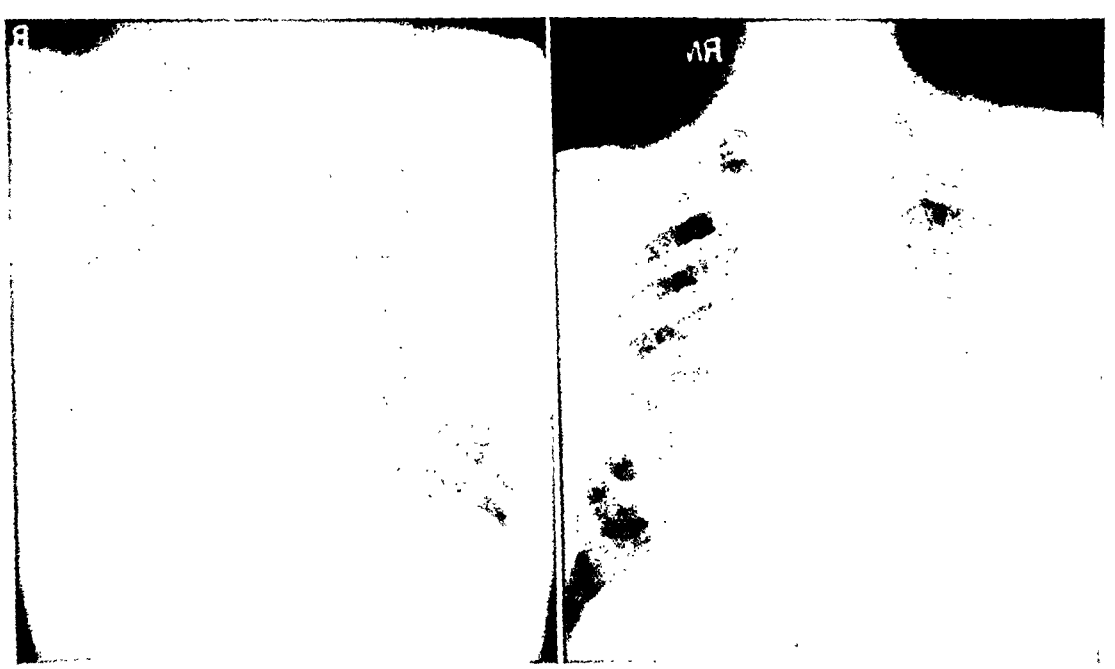


Fig. 1. Fig. 2.

Fig. 1. Roentgenogram showing Grade I visibility of right horizontal fissure, and enlargement of cardiac shadow.

Fig. 2. Roentgenogram showing Grade I visibility of right horizontal fissure. Two parallel lines are noted, indicating tangency of roentgen rays to fissure along two planes.

normalcy, namely, an increase in the intrafissural film of pleural fluid responsible for an x-ray shadow *in vivo*, but insignificant, neither cardiac nor pulmonary pathology, and the incidence of linear shadows corresponding to the right pleural fissures

TABLE I

Fissural Lines	Normal Series 501 X-rays	Tuberculosis Series 162 X-rays	Cardiac Series 283 X-rays
Absent	80%	59%	48%
Present	20%	41%	52%
Grade I	20%	30%	45%
Grade II	0.2%	7%	6%
Grade III	0%	4%	0.8%

This table shows the absence or presence and grade of lines corresponding to right superior fissure, in roentgenograms of the chest of normal control series, of tuberculosis series—including cases with bilateral or right unilateral infiltration—and of cardiac series of chest roentgenograms showing enlarged hearts.

TABLE II

Right Superior Fissural Lines	Normalcy	Effusion	Congestion
Absent—137 X-rays	86%	6%	8%
Grade I—130 X-rays	73%	14%	13%
Grade II—14 X-rays	14%	60%	26%
Grade III—2 X-rays	0%	100%	0%

This table shows the occurrence, in chest roentgenograms with enlargement of cardiac shadow, of signs of passive congestion, of pleural effusion, and of normalcy, excepting the presence of fissural shadows.

or absorbed at autopsy. The scarcity of satisfactory autopsy material has limited the solution of the problem in this direction (4).

Although occurrence of such linear shadows has been noted by Crecelius (4) in "almost 50 per cent of cases," their preponderance in x-rays demonstrating tuberculosis, or cardiac enlargement, appeared impressive to the author, and an analysis of this occurrence was undertaken in an effort to determine in a manner not yet employed in this problem the significance of the shadow under discussion. The presence of fissural shadows had been noted previously in heart disease (7, 13, 17) and especially in chronic passive pulmonary congestion, but its incidence in a normal control series had not been determined. Lines of exudative, or of proliferative origin, had also been reported relating to tuberculosis (12), to rheumatic fever (19), and to other types of inflammatory infiltration (14), but again without normal controls.

Accordingly, to determine the normal incidence of such linear projections and the effects upon their occurrence of inflammatory and hydrostatic factors—which presumably were the chief modifying influences—three groups of x-rays were selected and studied as follows: 501 chest roentgenograms were selected, showing was determined. One hundred sixty-two roentgenograms showing pulmonary tuberculosis involving the right lung, either alone or as a part of bilateral disease, were also examined for the incidence of these pleural lines. A third group of 382 roentgenograms in which the heart was definitely enlarged was reviewed, in a similar manner, the common denominator of cardiac enlargement being selected as the most dependable roentgenographic feature of cardiac disease and failure. Inspection of the hospital charts available in 211 of these cases demonstrated the dependability of this criterion. In all three series it was considered advisable to sub-group the linear shadows according to their fineness or coarseness, as suggested by Busi (2), to determine, if possible, any difference in significance of shadows of fine, medium, and coarse caliber. These calibers have been termed, respectively, Groups I, II, and III, and are illustrated by Figures 1, 3, and 4. Data relating to the incidence and caliber of the linear projections of the horizontal pulmonary fissure in these three series of x-rays are shown in Table I.

Examination of these data renders the pathologic significance of linear shadows of the right horizontal pleural fissure unquestionable, as well as the greater significance of the medium and coarse shadows over the finer projections. Thus, almost exactly one-fifth of otherwise normal chest roentgenograms showed linear shadows which in but one instance of 501 cases were of medium caliber, all the remaining pro-

jections being of the fine type and none of the coarse. Among the tuberculosis series, the incidence of lines was 100 per cent greater than normal, being found in 41 per cent of all roentgenograms, and with a definite shift toward the medium and coarse types of projection, which occurred in 7 per cent and 4 per cent of roentgenograms, respectively. Among the cardiac cases, the incidence of pleural lines was even higher, being 150 per cent greater than in the normal control series. Thus, 52 per cent of the roentgenograms of chests with cardiac enlargement showed the linear shadows, of which, however—as might have been expected lower percentages were of the medium and coarse types of lines than among the tuberculosis cases. These occurred in 6 per cent and 0.8 per cent of x-rays, respectively. Acknowledging the occurrence of linear pleural shadows in 20 per cent of the normal series, their significance in favor of pathology is thus unquestionable, with a notably greater incidence of lines in association with an enlarged heart than even with tuberculosis of the right lung.

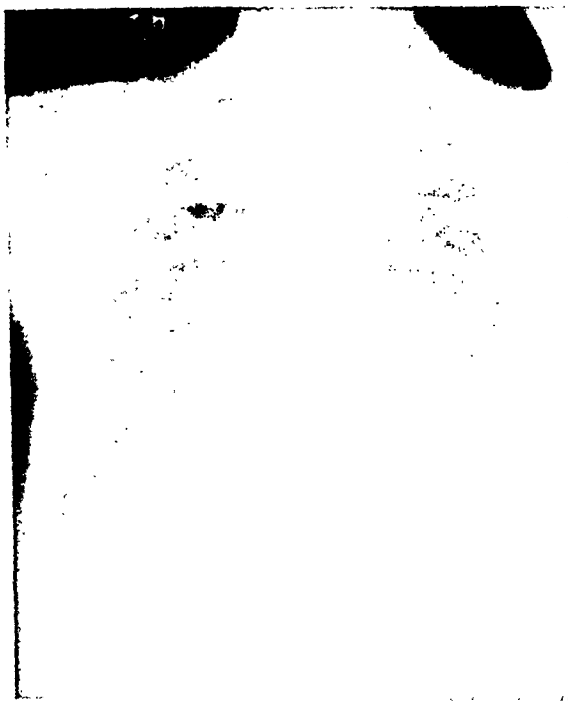


Fig. 3.

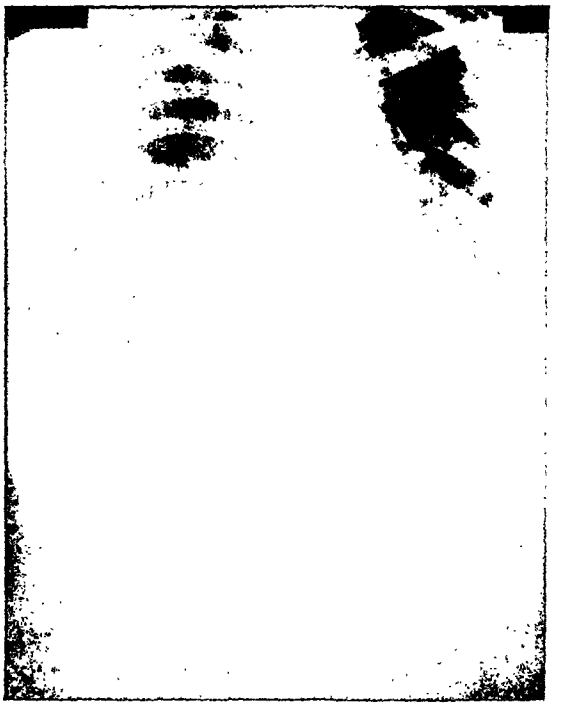


Fig. 4.

Fig. 3. Roentgenogram showing Grade II visibility of right horizontal fissure with enlargement of cardiac shadow.

Fig. 4. Roentgenogram showing Grade III visibility of right horizontal and oblique fissures due to accumulation of transudate, identified by transverse roentgenography, and disappearance with recovery of cardiac compensation.

This point determined, the question of the mechanism by which the opacity of the fissure was increased appeared important. Conceivably, the incidence of lines in the normal cases may represent the result of the fortuitous tangency of the interlobar pleura to the roentgen rays, a factor presumably constant in the tuberculosis and cardiac series. The 100 per cent increase in incidence of linear shadows in the tuberculosis cases might be due to inflammatory infiltration of the sub-pleural parenchyma of the lung, an inflammatory effusion into the fissure, or a pachypleuritis. The first and last would appear more likely to result in coarse lines, a conjecture compatible with the highest incidence of Grade III

lines in this series of roentgenograms. In the 150 per cent increase over normalcy in the occurrence of lines in the cardiac fifth showed slight generalized pleural effusion.

Twelve autopsies were available in cases

TABLE III

Type of Disease	All Heart Dis. 1933–1938	Line Absent 92 Cases	Line Grade I 103 Cases	Line Grade II 14 Cases	Line Grade III 2 Cases
Rheumatic	34%	14%	20%	14%	100%
Arteriosclerotic	16%	20%	19%	21%	0%
Hypertensive	23%	27%	32%	21%	0%
Syphilitic	3%	26%	18%	36%	0%

This table shows the percentage of occurrence of the principal types of heart disease in general cardiac hospital population, and in absence and grades of presence of lines in chest roentgenograms, corresponding to right superior pulmonary fissure.

cases, inflammatory factors are largely eliminated in favor of hydrostatic ones. This is reflected in the less frequent occurrence of heavy lines than in the tuberculosis series. Yet fluid might accumulate upon either the pulmonary or the fissural side of the pleura. Therefore, the determination of the incidence of frank pleural effusion elsewhere in the right chest and of the usual x-ray appearance of passive congestion appeared important. This analysis is noted in Table II.

The importance of pleural effusion in increasing the visibility of fissures is apparent from this analysis in the ascending incidence of frank effusion in the coarser types of linear shadows. The same observation pertains, in lesser degree, to passive congestion. Roentgenographs showing characteristics of both congestion and effusion were included with the group of effusions. Of the two mechanisms, however, hydrothorax would appear, from a consideration of the data tabulated, to be the more important factor.

This apparent importance of effusive and congestive factors, of proliferative changes in increasing the visibility of the horizontal fissure, and the pathologic significance of such visibility is further borne out by the autopsy material available from the cardiac series. Five autopsies were done on cases with enlarged hearts, but without demonstrable fissural lines in antemortem roentgenographs. Four of these showed entire normalcy of the right pleura and the absence of demonstrable effusion; the which had shown fine lines in antemortem x-rays. Of these, but one was normal, while seven showed right hydrothorax of varying degree and four had interlobar adhesions. Of the two autopsy reports available in material of medium caliber lines, one showed normalcy of the pleura with effusion, and the other both hydrothorax and adhesions. No autopsy was available on Grade III material. Again, therefore, the pathologic significance of pleural lines of even fine caliber is obvious, while the rôle of effusion in the creation of these shadows appears of greatest prominence. The type of heart disease represented in the autopsied cases included all the usually important classes without a significant preponderance of any one group.

It is further interesting to examine the 211 hospital charts, available in the cardiac series of roentgenograms, in terms of the types of heart disease represented. This analysis is made in Table III. Comparison is also made with the incidence of various types of heart disease among the cardiac population of the hospital in the years 1933–1938. The less common types of heart disease and the occasional unclassified cases are omitted because of the statistical insignificance of their numbers. The unusually low incidence of syphilitic heart disease in the general hospital population would appear to be due in part to discrimination in admissions, and, possibly, in part to early faulty classification.

Were the inflammatory factors of predominant importance in rendering fissural

pleuræ visible, as suggested by Starr and Parrish (18), the proportion of rheumatic heart disease to the degenerative types of heart disease in cases showing pleural lines should be relatively higher than in the cases showing no fissural lines. The incidences of rheumatic, arteriosclerotic, hypertensive, and syphilitic types of heart disease, however, were essentially the same in groups with and without the fissural shadows. It appears, therefore, that cardiac enlargement and presumably failure, with resulting effusion and passive congestion, are more important mechanisms in the visibility of pleural lines than any other factor common to a given type of heart disease. The lesser proportion of syphilitic heart disease in the general hospital population than among the group under discussion is possibly the result of factors noted heretofore, while the comparatively low incidence of rheumatic disease among the special groups under study, both with and without lines, is probably due to the large numbers seen before cardiac enlargement was significant, and, therefore, included among the hospital population but not admitted to the present study.

Among the instances of rheumatic disease in the present group, mitral valvular lesions predominated over aortic valvular lesions in the ratio of three to one, in that group demonstrating pleural lines, but occurred in equal numbers in instances in which pleural lines were not demonstrable. Among the mitral valvular lesions, insufficiency appeared in twice as many cases as did stenosis, judging from the clinical appraisal of the heart examination, regardless of the presence or absence of demonstrable x-ray fissural lines. It is thus apparent again that factors favorable to pulmonary congestion or pleural effusion predominate in those cases in which pleural lines are found.

Instances of retrogression of localized interlobar pleural effusions with recovery of cardiac decompensation have been reported (22), the localized accumulations being conditioned as already explained by incomplete obliteration of the pleural space. Such an accumulation is illustrated by Figure 4, in which the density in the right lung-field near the hilus is shown by lateral projection to be due to effusion in the oblique fissure adjacent to, and communicating with, the horizontal fissure, and disappearing with recovery of compensation, resulting in the ultimate disappearance of the line before the patient was discharged from the ward. The rôle of effusion in causing such linear visibility is thus again suggested.

SUMMARY

It would, therefore, appear to be unquestionable that although pleural lines corresponding to the right horizontal fissure, and at times or with special technic, to the oblique fissures of the lung, occur in approximately one-fifth of the normal chest roentgenograms, their 100 per cent greater incidence in cases with right-sided pulmonary tuberculosis, and their 150 per cent greater incidence in cases showing enlarged hearts are significant of their frequent pathologic character. Such pathology might conceivably include infiltration, mechanical congestion, and pleural effusion. It is demonstrated herein that all of these exert a definite influence when present. The predominant rôle of effusion into the pleural fissures with the creation of a film of fluid greater than normal, though still of capillary thickness, is evident, from a consideration of the above data, and this mechanism may be assumed to account for the increased visibility of the interlobar fissures, under the circumstances of the present study.

CONCLUSIONS

1. Lines corresponding to the horizontal fissure of the right lung occur in 20 per cent of normal chest roentgenograms.

2. The incidence of such lines in cases of right-sided pulmonary tuberculosis is 100 per cent greater than normal.

3. The incidence of such lines in cases with cardiac enlargement is 150 per cent greater than normal.

4. The lines of heavy caliber are particularly significant of pathology and rarely occur in otherwise normal chest roentgenograms.

5. The cause of such greater visibility of the horizontal fissure is most often a thickening of the film of interlobar pleural fluid by hydrothorax, of frequently otherwise undetectable degree.

6. Inflammatory pleural and subpleural condensations are occasional factors in increasing the visibility of these pleural lines.

7. Such increased visibility of the fissures occurs in cases with cardiac enlargement independently of the type of heart disease present, but particularly correlated with the degree of decompensation.

8. The significance of fine pleural lines in any given instance must be determined according to the merits of the individual case in view of the 20 per cent incidence of such lines in the series of otherwise normal roentgenograms of the chest.

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AN EXPERIMENTAL STUDY OF THE EFFECTS OF ROENTGEN RAYS ON THE GONADS OF THE SEXUALLY MATURE DOMESTIC FOWL[1]

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WITH the aid of standardized x-ray equipment we have undertaken a systematic study of the effects of roentgen rays on the gonads of the domestic fowl. In this study, we have attempted to correlate the age of the animal, the dosage given, and the results obtained. In the first part of the study (1) we were dealing with the embryo and the young chick. The present paper deals with the study of the adult male and female bird.

MATERIALS AND METHODS

The x-ray machine[2] used was mechanically rectified and provided with a Landauer roentgenometer. A universal Coolidge therapy tube was used. The set-up of the machine for the entire experiment was as follows: The kilowatt meter was set at 96, which delivered 112 kv.p. as measured by the sphere gap; the milliammeter was set at 6 ma.; the focal distance was 10 in.; the filter was equivalent to 4 mm. aluminum; the roentgenometer was kept at 3.2 micro-amperes, which, by calculation, gave 0.6 r per second. The desired r, or dosage, was obtained by varying the time of exposure.

Young, sexually mature White and Brown Leghorn hens and roosters were selected for the experiment. The age of the birds varied from eight months to one year. They were securely strapped onto a specially constructed stand with the back of the birds directed toward the target. The irradiation stand was so constructed as to facilitate adjustment of the focal distance for each separate bird.

All of the birds except the roosters of Series One were protected during irradiation by a sheet of lead. An opening 2 in. square was cut in the lead plate and placed directly over the gonad area during exposure to the x-rays. Series One was irradiated without such protection.

Both filtered and unfiltered rays were used in the present experiment. The former proved more effective and gave more steady results; for these reasons filtered rays were used almost exclusively.

Testicular tissue was fixed either in 10 per cent formalin and treated with mercuric acetate, or in Bouin's picric acid solution. The latter fluid proved also very satisfactory for ovarian tissues. The tissues were embedded in paraffin and sectioned from 4 to 10 micra in thickness. Hematoxylin-eosin staining was used.

RESULTS: IRRADIATION OF ROOSTERS

In this experiment, 71 specimens were used. Of these, 10 were controls. The 61 irradiated birds numbered 35 roosters and 26 hens. The experimental results obtained in irradiation of the males will be discussed under the following three topics.

Series One.—Ten roosters were irradiated with one dose of x-rays, ranging from 1,216 to 2,816 r. One week elapsed between irradiation and the time the bird was killed. Although the dorsal gonadal area was placed in direct line with the target the other areas of the body were not protected from the rays. In the lower ranges, namely, from 1,216 to 1,856 r, pycnosis of the nuclei in primary and secondary spermatocytes was the major visible pathology. There was a corresponding decrease in the number of spermatids. Spermatogonia and Sertoli cells seemingly were not affected. Likewise the interstitial tissue and vascular supply showed no alterations (Fig. 1). In the higher doses of this series, that is, from 2,176

[1] Submitted for publication in April, 1939.

[2] Type C model, made by the Standard X-ray Company.

to 2,816 r, pycnosis of the nuclei was accompanied by fragmentation of the cytoplasm. This was seen especially in primary and secondary spermatocytes, spermatids, and in some spermatogonia (Fig. 2). When the spermatids possessed pycnotic nuclei, the spermatozoa were absent in the lumina of the tubules. The interstitial tissue exhib-

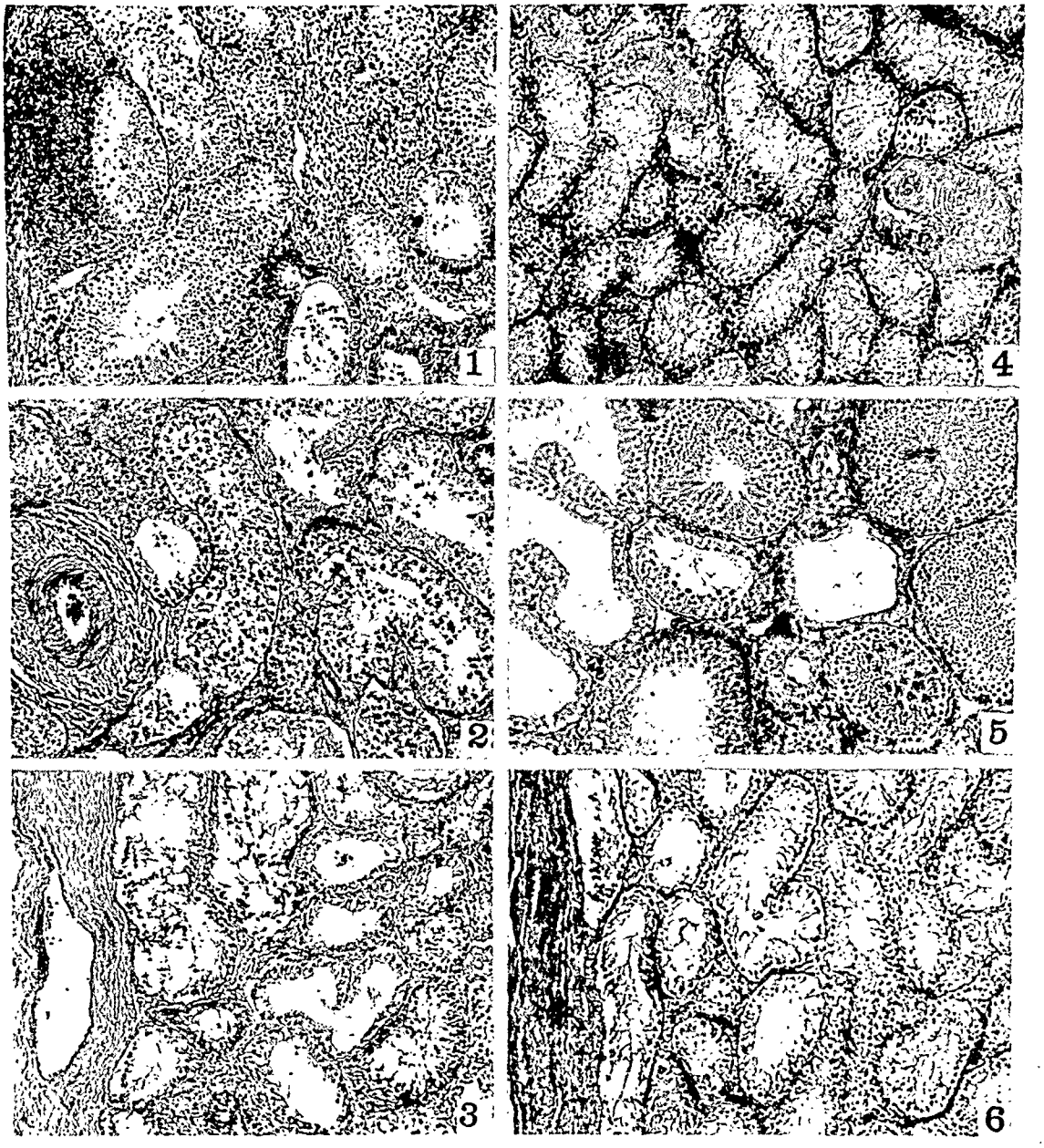


Fig. 1. Section of a testis irradiated with 1,536 r. Apparently normal spermatogenesis occurs in most of the tubules. Note infiltration of lymph cells (× 125).

Fig. 2. Section of a testis treated with 2,496 r. *Destruction and exfoliation of seminiferous epithelium*; thickening of adventitial coat of arteries (× 125).

Fig. 3. Section of a testis x-rayed with 2,496 r. Destruction of seminiferous epithelium more advanced. Note thickened tunica albuginea (× 125).

Fig. 4. Section of a testis irradiated with 2,496 r. The breaking down of the Sertoli cell and the spermatogonia is shown. The arteries are practically obliterated (× 125).

Fig. 5. Section of a testis treated with a cumulative dosage of 400, 600, and 800 r. "Denuded" tubules occur side by side with tubules with apparently normal spermatogenesis (× 125).

Fig. 6. Section of a testis treated with a cumulative dosage of 1,000, 1,200, and 1,400 r. The destruction is similar to that seen in Figure 4 (× 125).

ited no microscopic changes but there was an increase in the thickness of the adventitial coat of the small arteries. Some evidence of an endarteritis was present in some of these vessels.

Series Two.—Twelve roosters were irradiated with doses ranging from 2,176 to 2,816 r. These specimens were permitted to live three weeks after irradiation. Histologic examination revealed that spermatogonia and Sertoli cells showed a greater resistance to the action of the x-rays. Spermatocytes were, in general, necrotic, also characterized by pycnosis and plasmolysis (Fig. 3). Spermatids as well as spermatozoa were absent in the tubules. The interstitial tissue seemingly was of normal structure. The arterioles exhibited an endarteritis (not obliterative), and a thickening of the tunica adventitia. In all of the specimens of this series the tunica albuginea showed an appreciable increase in thickness. In some instances there was as much as three times the thickness found in the controls. No. 122 of this series (Fig. 4), which received 2,496 r of filtered rays, deserves special mention: in this, the tubules were filled with the broken-down substance of the Sertoli cells, and only a few of the spermatogonia were present. Other findings of this specimen corresponded to those of the other members of this series.

Series Three.—It was composed of 13 roosters. These specimens received three successive doses of x-rays at intervals of one week and were killed three weeks after the last irradiation. The dosage varied from the low combination of 400, 600, and 800 r to the maximum of 1,200, 1,400, and 1,600 r. Histologic sections, viewed macroscopically, presented a mottled appearance. Microscopically the mottling was seen to be due to vastly different changes in adjacent tubules. In many tubules, the seminiferous epithelium consisted of but a single layer of spermatogonia, whereas the neighboring tubules possessed all the layers of normal seminiferous epithelium with an apparently increased mitotic activity and spermatogenesis (Fig. 5). The proportion of "denuded" tubules to unaffected tubules increased as the dosage increased. In specimens having received 1,000 r or more as an initial dose, the seminiferous epithelium was reduced to a single layer of cells, spermatogonia and Sertoli cells. These cells showed definite signs of degeneration (Fig. 6). The adventitia of the arteries and the tunica albuginea of these specimens were much thicker than in the controls. Accumulations of lymph cells were found in practically all of the x-rayed material. No definite alterations could be demonstrated in the interstitial tissue.

IRRADIATION OF HENS

Just as the testes showed structural changes due to irradiation, the ovaries also demonstrated the effects of x-rays. Altogether, 20 hens were subjected to single doses of x-rays. The doses ranged from 400 to 2,176 r and the hens were killed three weeks after irradiation. The mortality rate was very high in the specimens treated with 1,856 r or more and, therefore, only five specimens are present in this lethal dosage range.

Low Dosage Series.—The specimens irradiated with 400 to 700 r showed initial injury to the ovary which was evidenced by the shrinkage of the nucleus of the ovum (Fig. 7). Extensive follicular degeneration was noted with dosages ranging from 800 to 1,200 r. The yolk of many large follicles was invaded by proliferations of the cells from the stratum granulosum and in some instances by connective tissue from the theca folliculi. The effects of irradiation of small follicles were characterized by the scalloping of the nuclear membrane of the ovum and the atrophy of nuclear and cytoplasmic contents. The germinal epithelium presented considerable variability in the same specimen. In some parts of the ovary it appeared normal in structure, whereas in most parts it consisted of a single layer of flattened cells. Primary follicles were found only in regions of active germinal epithelium. Hemorrhagic areas were found in many parts of the ovary but particularly in the cortex,

where they showed signs of degeneration and absorption. Multinucleated giant cells were abundant in such areas. A variable amount of pigment granules, resembling melanin, was present in the interstices of the cortical tissues. The medullary por-

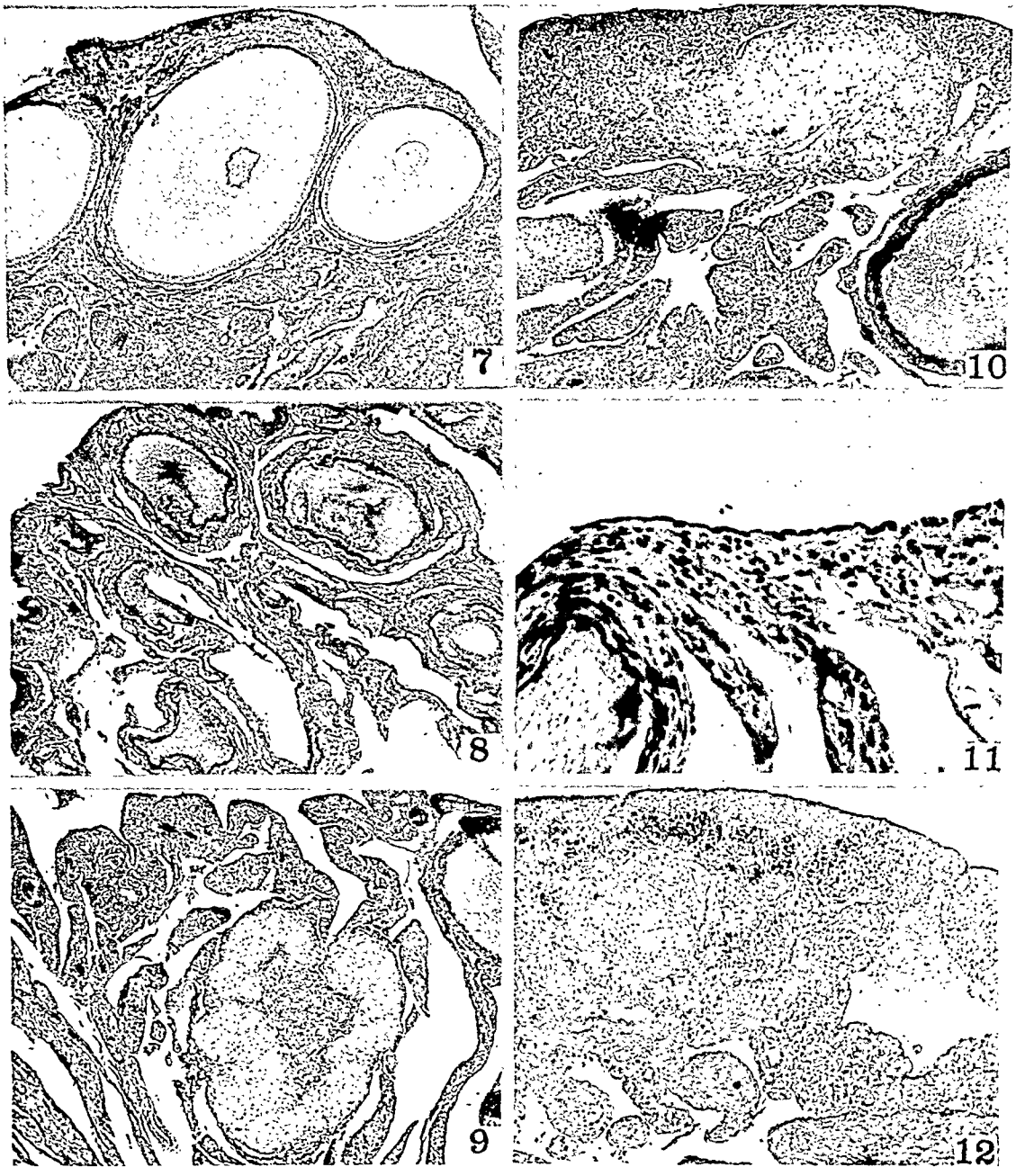


Fig. 7. Section of an ovary showing initial injury caused by 400 r of x-rays. Note the shrinkage of the nucleus and the scalloping of the nuclear membrane (× 83).

Fig. 8. Section of an ovary irradiated with 2,176 r. Note destruction of follicles and germinal epithelium (× 90).

Figs. 9 and 10. Sections from an ovary x-rayed with 2,176 r showing different stages of degeneration of the ovarian follicle (× 420).

Fig. 11. Section of an ovary irradiated with 2,176 r. Note the exfoliation of the germinal epithelium (× 420).

Fig. 12. Section of an ovary treated with a cumulative dose of x-rays. Note the melanin-like pigment granules (× 150).

tion of the ovary presented the least pathology.

High Dosage Series.—The results obtained from hens irradiated with dosages of from 1,200 to 2,176 r differ from those of lower dosages in severity of injuries to the ovary. The follicular degeneration was greater, as indicated by ingrowths of the cellular layer and connective tissue into even the small follicles. The degenerating follicle seldom remained spherical in shape. With the atrophy of the ovular contents the entire follicle collapsed. The most common form of a collapsed follicle was that of a crescent (Fig. 8). Gradually the contents of the follicle, except some of its connective tissue, disappeared entirely (Figs. 9 and 10). The germinal epithelium was more severely affected than in the specimens of the former group. Mitosis was nowhere seen; instead, exfoliation of its cells was taking place in all parts of the ovarian cortex. In many places the ovary was covered by a thin layer of connective tissue (Fig. 11). As would be expected, primary follicles were totally absent from ovaries treated to higher dosages of x-rays. Pigment granules and hemorrhagic areas were noted in the cortex. The thickening of walls of small arteries and the reduction of size of their lumina were found in practically all of the ovaries irradiated. In some instances, the lumina were almost occluded.

Cumulative Dosage Series.—A small series of hens received three consecutive doses of filtered x-rays at intervals of one week. The dosage varied from the low combination of 400, 600, and 800 r to the maximum of 1,200, 1,400, and 1,600 r. The hens were killed three weeks after the last irradiation. The results obtained were fairly uniform in all the specimens of this series. Almost all follicles, large and small, showed degenerative changes. In large follicles, the nuclei were in the last stages of disintegration and the yolk was encroached upon by proliferations of the cellular layer of the follicle, and in some instances by connective tissue. Small follicles invariably possessed scalloped nuclei and in many instances were being obliterated by connective tissue. Primary follicles were not found in any of these specimens. Perhaps the greatest variability in the ovaries of this series was noticeable in the germinal epithelium, particularly in the specimens that had received the low dosage combinations of x-rays. In most parts of the ovary it was reduced to a single layer of flattened cells; rarely it was totally destroyed and exfoliated. In some places what appeared to be an active germinal epithelium was found. The cells were cuboidal in shape with oval or spherical nuclei, and indications of mitosis seemed to be present. However, fully formed germ cells could not be found any more than primary follicles. In the higher dosage series, the germinal epithelium was reduced to a single layer of flattened cells or was totally destroyed and exfoliated. There were no signs of regeneration. In all of the ovaries of this group, hemorrhagic areas containing multinucleated giant cells were found. A variable amount of pigment, morphologically similar to that described in a series treated with a single dose of x-rays, was present in the stroma of the cortex (Fig. 12). The medulla of these ovaries showed increase of fibrous connective tissue, and the arterioles had thickened walls and small, sometimes almost occluded, lumina.

DISCUSSION

With few exceptions the results of the present experiment parallel the results of the first study in which embryos and young chicks formed the material (1). One of the major exceptions of this experiment consists in the absence of cell-cord formation in the ovaries injured by x-rays; growing and fully formed ovarian follicles degenerate completely, primary follicles, very likely, follow the same fate, although the survival of individual cells of the broken-down follicle is not excluded. Another of the major exceptions is that no increase of interstitial tissue has been noted in the present experiment. The dosage required for initial injury was

higher in this than in the former experiment. The testes proved to be more resistant to x-rays as in the former experiment.

An x-ray dosage of 1,200 r was required to produce initial injuries to the seminiferous epithelium of the adult male bird. The first cells injured were the rapidly multiplying primary spermatocytes and next the secondary spermatocytes. The spermatogonia, spermatids, and Sertoli cells seemed to be affected the least. Similar observations have been made in rats and guinea pigs (2). In the more severely affected seminiferous tubules, there was a total lack of primary and secondary spermatocytes; yet, the transformation of spermatids into spermatogonia appeared undisturbed as far as the microscope could reveal. The injuries produced by 1,200 or more units of x-ray varied with different animals and with different levels of the same seminiferous tubule.

The first of the so-called denuded seminiferous tubules occurred in testes treated with dosages of about 2,000 r. In such tubules all of the seminiferous epithelium has been destroyed except the spermatogonia and the Sertoli cells. The cellular débris has been discharged or absorbed and the tubules appear empty in sectional views. The number of such denuded tubules varied with the dosage used; in testes treated with 2,400 to 2,800 r, practically all the seminiferous tubules were denuded. The spermatogonia and Sertoli cells that remained attached to the basement membrane of the tubule were few in number and abnormal in appearance. It seems likely that with higher dosages, all of the seminiferous epithelium is permanently destroyed. This conviction is supported by the findings in the specimens killed three weeks after irradiation when there was ample time for recovery. The increased amount of connective tissue in the tunica albuginea and the adventitia of the arteries seems to indicate stimulating effect of x-rays on supporting tissues.

In the cumulative series, the denuded tubules varied with the total dosage. There were few of the denuded tubules with 1,800 r, more with 3,600 r, but with the total dosage of 4,200 r they were the only tubules present. The histologic structures of the seminiferous tubules, tunica albuginea, and the arterial coats of the high cumulative dosages are identical with the same structures of the testes which received a high (2,800 r) single dose of x-rays. It is clear that considerable acclimatization has taken place in cumulative irradiation. The occurrence of a denuded portion of tubules aside from those with normal spermatogenesis is interpreted as a function of the cyclical activity of the seminiferous tubule. The portions of tubules in a low cyclical phase at the time of irradiation have escaped damage, whereas those in high activity have been destroyed.

The wrinkling or scalloping of the nuclear membrane proved to be the first observable sign of x-ray injury to the ovary. It occurred in all irradiated ovaries, beginning with a dosage of 400 r. Whether or not recovery follows such initial injuries cannot be answered from the histologic observation. X-ray injuries of more serious nature were first noted in specimens irradiated with 800 r, and increased with the increase of the dosage. Most commonly, such follicles collapse into crescent- or star-shaped forms. The collapse is probably due to shrinkage of the nucleus and the yolk. The ingrowing stratum granulosum and theca folliculi seem to assist in the resorption of the follicular contents. After the yolk is removed the cells are large, polyhedral, light-staining structures that have some resemblance to luteal cells of mammals (Fig. 9). Similar structures have been observed in the follicles of x-rayed mice (2, 3, and 4). It is likely that total destruction of the follicular content of the ovary is produced by dosages ranging from 1,800 to 2,200 r.

The total dosage in the cumulative series of hens was as high as in the case of roosters, *i.e.*, from 1,800 to 4,200 r. This is remarkable when it is remembered that the lethal dose of hens was in the neighborhood of 2,200 r, whereas that of roosters was

about 2,900 r. From these data it would appear that the power of acclimatization to x-rays is greater in female than in male birds.

The reaction of the germinal epithelium to the effects of x-ray brings to light some interesting facts. It is effected with dosages as low as 400 r but the effect is not the same in all parts of the ovary. It is reduced at some places to a single layer of flat cells, whereas in other places it consists of several layers of cells in thickness. In the former location there are no signs of mitosis or primary follicles, whereas in the other location both mitosis and primary follicles occur. Mitosis and primary follicles have been noted in specimens treated with a single dose of 1,200 r. Beyond that dosage complete destruction probably results, which is witnessed by cellular exfoliation. The findings are the same with cumulative dosages. It is clear that the germinal epithelium acts in a cyclical manner, the same as the seminiferous epithelium. This conclusion is further strengthened by the fact that in the cumulative dosages the areas of activity decrease as the dosage increases. This observation, as far as the writers know, has not been reported before.

The melanin-like pigment granules were found in both testes and ovaries. They were, however, by far more abundant in the ovaries than in the testes and particularly in the ovaries treated to cumulative dosages. Their sources of origin remain unknown but the frequency at which these granules occur in irradiated ovaries and testes forces the conviction that x-rays are the causative factors.

SUMMARY

In the present experiment 71 young, sexually mature birds were used. Of these there were five male and five female controls, 35 male and 26 female experimental birds. Twenty-two of the 35 males were irradiated with a single dosage ranging from 1,276 to 2,816 r; 13 received three cumulative dosages, the total of which varied from 1,800 to 4,200 r. Twenty of the 26 hens were treated with a single dose, ranging from 400 to 2,176 r, and six were irradiated with three cumulative doses, the total of which varied from 1,800 to 4,200 r. The roosters of Series One were sacrificed one week after irradiation, and the rest of the experimental birds were killed three weeks after treatment. The results are as follows:

(1) With few exceptions the results of the present experiment parallel those of the former study.

(2) A dosage of 1,276 r was required for initial injury and 2,400 to 2,800 r for total destruction of the seminiferous epithelium.

(3) The order of susceptibility of the cells of the seminiferous epithelium to x-rays is: spermatocytes, spermatids, spermatogonia, and Sertoli cells.

(4) X-ray injury to the seminiferous tubule varies with the level of the spermatogenic wave; the injuries are least at levels of least activity and most at levels of active cellular proliferation.

(5) Initial injury to the ovarian follicle was produced by 400 r, follicular disintegration by 800 r, and total destruction of the follicles from 1,800 to 2,200 r.

(6) The destruction of the ovum is aided by the cells of stratum granulosum and theca folliculi.

(7) Initial injury to the germinal epithelium occurs with 400 r, total destruction with 1,200 r.

(8) The fact that areas of total destruction and areas of cellular proliferation occur simultaneously in the same irradiated ovary proves the presence of cyclical activity in the germinal epithelium.

(9) Acclimatization to the effects of cumulative doses of x-rays was noted in both testes and ovaries of treated birds.

(10) Hyperplasia of fibrous connective tissue was noted in the walls of small arteries and medullary stroma of the ovary and in the walls of small arteries and tunica albuginea of testes.

(11) No definite changes due to x-rays could be demonstrated in the interstitial tissue of the gonads of either sex.

(12) A brown melanin-like pigment was

found in variable quantities in the stroma of irradiated ovaries and testes.

(13) The gonads of birds appear to be more resistant to the effects of x-rays than are the gonads of mammals.

(14) Individual variation was much in evidence in the experiment.

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CASE REPORTS

RETOTHEL SARCOMA OF THE STOMACH

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According to Edling (1), in 1914, Ewing proposed the term "reticular-cell sarcoma" for a portion of the group of tumors then being referred to collectively as "lymphosarcoma" and characterized by an origin not from the lymphatic cells of lymphoid tissue but from its reticulum cells.

The term "retotheliosarcoma," applied to the same group of tumors, has been current since it was introduced by Roulet, of Berlin, in 1930, in his description of certain tumors of lymph nodes and of the epipharynx, tonsils, and other sites in the upper respiratory tract. The further usage "retothel" is merely a variant.

With relation to the larger parent group of sarcomas, leaving out the adult and differentiated forms, lymphosarcomas form one member of a chain of which the other two members are spindle-cell sarcomas and round-cell sarcomas (2). Lymphosarcomas are the most numerous and important of the three, so far as sarcomas in the stomach are concerned.

For a critical discussion of the pathology, the reader is referred to the excellent article of Edling (1). The latter says, in summing up: "*I have adopted the term proposed by Ewing* because it seems to me to define the nature of the growth more accurately than the name of 'lymphosarcoma' which is more commonly employed."



Fig. 1.

The literature contains less than 400 cases of recticular-cell sarcoma of the stomach found at operation and autopsy (3). The lesion was first described by Morgagni, in 1750. Of the cases found at operation, 32 per cent are living after a period of years (3). These figures are not in agreement with those of Haggard, who found a 64 per cent survival after a period of years.

Balfour and McCann reported the operability of the lesions as being 66 per cent of the cases explored.

The youngest patient to have a reticular-cell sarcoma of the stomach was three and one-half years of age (3).

The relative incidence of this lesion, as compared to carcinoma, is usually estimated at about 1 per cent (5). Balfour and McCann found it to vary from 1:56 to 1:226 (6).

Since the constantly changing pathologic concept of the lymphoblastomas brings with recurring insistence a consideration of the blood picture, it may be stated that so far as lymphosarcoma (or reticular-cell sarcoma, or retotheliosarcoma or retothel sarcoma) is concerned, there is nothing constant about the blood picture. Wiseman (7) concludes that "lymphosarcoma is a disease in which there is a transformation of the lymphocytic strain of cells . . . it may show a secondary anemia, or a leukemic or sub-leukemic blood picture."

When the latter is pronounced, the term "leukosarcoma" is frequently employed. Lymphosarcoma may exist in the presence of a full-blown leukemia (13).

The clinical appraisal of sarcoma of the stomach does not extend very far back into our surgical records. As late as 1910, Aimé Guinard (4) questioned the malignancy of sarcoma of the stomach and classed it doubtfully in a group of benign tumors in his *Grand Traité de Chirurgie.*

There is nothing constant about the symptomatology of this lesion. It has been linked with a history of trauma by a few (8 and 9). In Clar's case, the patient, a man 22 years of age, was knocked to the pavement by an automobile, most of the force of the blow being received in the epigastrium. Five months later he was admitted to the hospital with stomach complaints, and after an x-ray examination had shown a filling defect diagnosed as carcinoma, operation revealed a lymphosarcoma.

Jones and Carmody (9) tell of a boy, nine years of age, who was struck in the abdomen by a baseball. His stomach symptoms developed following this injury, and a lymphosarcoma was found at operation. Following resection, the authors obtained a permanent cure (19 years). These writers are impressed by the number of five-year cures they have discovered, and believe that, in selected cases, surgery gives an excellent prognosis.

Others (2) have been struck with the response of the lesion to x-ray therapy.

One case has been reported with a duration of five years before coming to operation (10), in which the clinical symptoms strongly suggested duodenal ulcer. In this case, incidentally, the x-ray examination was relatively negative. Hunt operated upon a case occurring in a child of three and one-half years, who had pyloric obstruction and a mass; post-operative recovery appeared to be complete (3).

Stomach symptoms may be altogether absent (11) and there may be only intense anemia, optic neuritis, and wasting.

The roentgenogram is not in the least diagnostic, so far as naming the lesion to be lymphosarcoma is concerned. There is usually a filling defect in the stomach, which, for obvious reasons, is going to be diagnosed carcinoma.

Golden (12) believes that if there is anything constant about the roentgenogram, it may be a filling defect in which an ulcer crater is made out: this in a young person might lead to a suspicion of sarcoma.

Our patient was a male, A. K., aged 32, of Polish descent. For five months before his admission to Cook County Hospital he had been complaining of a dull, epigastric pain, having no relation to food and shifting once in a while to the left loin. The pain usually came on in the forenoon and lasted for a few hours. It was not severe, and was of an aching character. For two to three days at a time he would have complete relief from the pain; later in the course of the disease it became unremitting.

He had never vomited; he had lost about twenty pounds in weight.

His gastric analysis showed no free acid, combined 9, total 9; there was a small amount of blood and mucus present. Subsequent examinations showed large amounts of blood several times.

Non-protein nitrogen was 33; serum amylase was 60; Wassermann, negative. Blood picture showed hemoglobin, 70 per cent; red blood cells, 4,410,000; white blood cells, 5,500; polymorphonuclears, 66 per cent; lymphocytes, 24 per cent; monocytes, 8 per cent; basophils, 0; eosinophiles, 2 per cent.

On admission, and as a preliminary diagnosis, acute diverticulitis of the colon and acute pancreatitis were considered; hydronephrosis was also considered.

The patient was sent for x-ray examination of the gastro-intestinal tract. Fluoroscopically, and on films, there was a large irregular filling defect, involving both the pars media and pars pylorica of the stomach, which was reported as follows: "Infiltration of the pars media and pars pylorica of the stomach characteristic of an advanced neoplastic process."

Though the films hardly suggested resectability, it was decided to give the patient the benefit of an exploration; this was done on Dec. 17, 1938.

The description of the gross pathology by the surgeon was as follows: "There is a hard mass on the greater curvature of the stomach near the pars media surrounded by massive hard glands and many glands are in the peritoneum and omentum. Five hundred cubic centimeters of peritoneal fluid were removed."

Nothing could be done surgically and the incision was closed. Later, the patient was sent for x-ray therapy, which he received until the time of his death, Jan. 6, 1939.

A gland near the stomach was removed at operation, and the biopsy report is as follows: "Biopsy of lymph node reveals an active retothel sarcoma."

SUMMARY

A case of retothel sarcoma of the stomach is reported, having the following characteristics:

1. The upper abdominal pain of which the patient complained suggested particularly neither ulcer nor carcinoma; on the contrary the tentative working diagnosis included diverticulitis of the colon, pancreatitis, and hydronephrosis.
2. Results of Ewald meal were similar to those in carcinoma.
3. The roentgen appearance of the stomach after barium meal most suggested carcinoma.
4. The patient lived approximately six months after the appearance of his first symptoms.

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THE MODERN HOSPITAL AND ITS RELATION TO THE PRACTICE OF MEDICINE

During the past twenty or thirty years, there have been rapid and extensive changes in the traditional function of the hospital in its relation to the practice of medicine and the delivery of medical care to the American people.

A much larger portion of sick patients are cared for in the private general hospital to-day than was the case a generation ago. Out of every dollar spent for medical care in this country, approximately twenty-four cents goes to pay a hospital bill, either in private or government hospitals (1). This represents a cost of about 656 million dollars per year, of which 300 millions is provided by taxes, 302 millions by patients' fees, and the remainder by contributions and endowments (2). The costs of hospitalization, as a result of illness, is almost as high as the total cost for private physicians' services during a given year. About 86 per cent of the total families in the United States require the services of a physician in an average year but only 20 per cent of the total have at least one hospital case (3). Less than 50 per cent of all family illnesses are hospitalized, yet the costs for hospitalization alone in these cases equals the entire expenditure for all illnesses of the entire family in an average year (4). Half the family bills for illness goes for hospital care, but in any one year only one person in fifteen has hospital care (5).

The cost of medical care in America, therefore, is very largely a cost for hospital care. That this latter cost is entirely justified, is a matter open to serious question. A number of factors have contributed to the increased use of hospitalization and the consequent increased cost of sickness. Many qualified students of the question declare that there is too much hospitalization, and that many patients subjected to the expensive facilities of the hospital could well be cared for in their own home. Three factors are responsible for this alleged over-hospitalization: doctors have referred their patients to the hospital because it was more convenient for the doctor to render his services there; patients have demanded that they be admitted to the hospital when they could just as easily have been treated in their homes, and hospitals have encouraged the use of their facilities with an occasional disregard of the need of the patient for those facilities.

Whether deliberately or not, current tendencies on the part of hospitals find their keynote in the first recommendation made by the Committee on the Costs of Medical Care. In 1929, the Committee recommended that medical care be furnished by groups, organized around a hospital (6). Thus the hospital becomes the dominant factor in the delivery of medical services. The importance of the traditional family doctor as the prime figure in an adequate health program is obscured in a trend toward institutionalized medicine with the hospital as the central figure.

Steps toward this goal were made when hospitals began the operation of pay clinics and out-patient departments in competition with the private physician's office. Abandoned was the old concept that out-patient services and part-pay clinics should be maintained only in teaching institutions where patients should be used for clinical material. Further steps were taken when the "middle rate" plan was adopted by a number of hospitals, in which patients were admitted on an adjusted fee basis including not only the hospital bill, but the bill for medical care as well. Other hospitals instituted "flat rate" or "all inclusive" plans under which a patient could be admitted to the hospital for diagnostic services and pay a flat fee covering the complete cost for medical services and hospital facilities. Still further and more radical steps were taken when hospitals began the sale of insurance plans which offered as benefits not only the use of the physical facilities of the hospital, but the services of medical specialists as well.

Income from endowments and contributions was sharply curtailed after 1929. The 7,000 hospitals in this country represent a capital investment of more than 3 billion dollars, with about 22 millions added each year for expansion (7) and the interest on these investments plus other fixed charges created a difficult problem in finance. Hospitals began to cast about for some new source of revenue to take the place of decreased endowment income.

Group hospitalization insurance presented

itself as a happy solution for the problem, insofar as hospitals were concerned. Started as an experiment by one hospital in Dallas, in 1927, the idea gained little attention until hospitals began to feel the pinch of depression. Then it began a wide and rapid growth.

Less than 65 per cent of the beds in non-governmental hospitals are normally occupied (8), yet fixed charges remain about the same regardless of the number of occupants. So even if hospital insurance increased the number of patients, the steady annual income from insurance premiums would provide the new and necessary revenue.

If hospitals had stopped with the establishment of insurance plans for hospitalization, there would have been little objection from the medical profession which has guarded so zealously the traditional system of American medicine. But, unfortunately, a number of hospitals went still farther to secure new revenue. Ostensibly operated on a non-profit basis, they began to adopt the rôle of a middle-man in the delivery of certain medical services to realize a profit on the services of physicians. Out-patient clinics for pay patients began to grow. Hospitals began to compete with private physicians' offices. Pathologists and radiologists working in the hospital found themselves competing with themselves in their private offices and with their colleagues. Frequently the department of radiology or pathology was operated in such a manner that a large portion of the fees patients paid for these medical services were diverted into the hospital treasury to meet the interest on building investments and other charges.

Here marks a radical departure in the former position of the private non-profit hospital in the medical picture. Doctors had always regarded the hospital as a specially equipped hotel where patients, who could not be satisfactorily treated in their own homes, could be hospitalized. Now the hospital was beginning an invasion of the medical field, and assuming the position of a commercial entrepreneur by interjecting itself between a doctor and his patient, demanding its portion of the fees passing between the patient and his doctor. Many hospitals collected premiums under insurance contracts providing not only hospitalization but the services of physician, radiologist, and pathologist as well.

So the lines of difference were clearly drawn. The medical profession has come to the realization that further encroachments on all types of medical practice are almost sure to follow unless hospitals can be prevailed upon to abandon this unfortunate course, return to the business of hospitalization, and leave the practice of medicine to private physicians.

The medical profession has long maintained that the institutionalization of medical practice or the application of the principles of socialization would result in a lowering of the quality of medical care available to the American people. Taking cognizance of the gradual trend toward such a condition through encroachment of hospitals upon the field of private medical practice, the Judicial Council of the American Medical Association at the annual meeting in 1936, issued a strong statement to the effect that:

> ". . . It would seem that in this time of extensive changes in hospital economics the point had arrived at which further marriages between hospitals and staff physicians that make the doctor of medicine the servant of the hospital should be stopped and a series of attempts at divorce among marriages that have already taken place should be instituted. Our accepted ethical principles are adequate at the present time and hospitals would be of invaluable assistance. It is not an impossible task but will need a militant local and national ethical spirit behind it and a frowning on those individuals in the profession who on personal grounds do not object to the gradual subjugation of the medical profession in the growth of hospital domination" (9).

The Code of Ethics of the A.M.A. provides that:

> "It is unprofessional for a physician to dispose of his professional attainments or services to any lay body, organization, group of individuals, by whatever name called, or however organized, under terms or conditions which permit a direct profit from the fees, salary, or compensation received to accrue to the lay body or individual employing him. Such a procedure is beneath the dignity of professional practice, is unfair competition with the profession at large, is harmful alike to the profession of medicine and the welfare of the people, and is against sound public policy" (10).

In this manner the conflicting viewpoints of the medical profession and the hospital group are defined.

The fears of the medical profession in this regard are fully justified by recent statements appearing in hospital publications. These statements evidence a complete disregard of the desires and recommendations of the medical profession. Repeatedly, statements have appeared that hospital service includes radiologic, anesthesiologic, and laboratory services; that the hospital depends upon the profits from the operation of certain medical departments to

maintain the hospital and to increase its income; that the services of the clinician, the surgeon, the pathologist, and the radiologist, as well as the nurse, are a part of the hospital and are services to be provided by the hospital, free from the dictation of medical men.

Practically all States in the Union have enacted statutes prohibiting the practice of medicine by corporations. Similar statutes prevail prohibiting the corporate practice of law, dentistry, and other learned professions. These laws have been passed for the simple reason that the practice of the learned professions by artificial legal entities would not be to the best interest or welfare of the people. Hospital corporations are artificial entities and it is hard to understand why the same reasoning should not be applied in their case. The public would surely suffer as the result of this kind of corporate practice the same as it would in the case of non-hospital corporations. The type of organization is relatively immaterial. In any case, the traditional and indispensable personal relationship between a doctor and his patient will be destroyed or injured under such conditions.

It is true that the authority of the common law is not clear upon the question of the corporate practice of medicine by hospitals. Although important decisions have been rendered by the Supreme Court in several States upholding the statute prohibiting corporate practice, there seems to be a universal reluctance to apply such rulings to hospital corporations. The law, however, has traditionally lagged at least a generation behind the sciences, and the question involved here is a scientific and not a legal one. The important point to consider is whether or not the science of medicine will be preserved and advanced. The general opinion among medical men is that corporate practice, either by hospitals or any other kind of corporation, will hinder the progress of the science and practice of medicine.

The medical profession has found it necessary to wage a militant battle against various forces which have, during recent years, attempted to override the opinion of medical men and bring about revolutionary changes in the form of medical practice in America. In the past, the medical profession has looked upon the hospital as its partner and ally in a relentless fight to preserve the practice of medicine from prejudicial influences.

It will be a great misfortune if the unselfish demands of the medical profession, standing on a firm realization of idealistic principles and scientific truths, be disregarded by a former ally and friend and the hospital shall be found on the side of those dangerous agencies which would substitute their opinions, regarding matters of health, for the opinions of the doctor.

The threat of hospitals in invading the field of medicine, upsetting the traditional relationships between physician and patient, and supplanting free private enterprise with regimented institutionalization is no less than the threat of other radical forces which the hospitals themselves have denounced.

MAC F. CAHAL
Executive Secretary

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RADIOLOGICAL SOCIETIES IN THE UNITED STATES

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Radiological Society of the Academy of Medicine (Cincinnati Roentgenologists).—*President,* Archie Fine, M.D., 707 Race St., Cincinnati; *Secretary-Treasurer,* Justin E. McCarthy, M.D., 707 Race St., Cincinnati, Ohio. Meetings held third Tuesday of each month.

PENNSYLVANIA

Pennsylvania Radiological Society.—President, Louis A. Milkman, M.D., Medical Arts Bldg., Scranton; *First Vice-president,* James E. Ginter, M.D., Dubois; *Second Vice-president,* Alexander Stewart, M.D., Shippensburg; *Secretary-Treasurer,* L. E. Wurster, M.D., 416 Pine St., Williamsport; *President-elect,* Harvey N. Mawhinney, M.D., 6546 Darlington Road, Pittsburgh; *Editor,* William E. Reiley, M.D., Clearfield; *Assistant Editor,* Sydney J. Hawley, M.D., Danville. Next annual meeting to be held May 17 and 18, 1940, at Hershey Hotel, Hershey, Pa.

The Philadelphia Roentgen Ray Society.—President, Joseph E. Roberts, Jr., M.D., 403 Cooper St., Camden, N. J.; *Vice-president,* Jacob H. Vastine, M.D., Medical Arts Bldg., Philadelphia; *Secretary,* Barton R. Young, M.D., Temple University Hospital, Philadelphia; *Treasurer,* Fay K. Alexander, M.D., Chestnut Hill Hospital, Philadelphia. Meetings held first Thursday of each month at 8:15 P.M., from October to May in Thomson Hall, College of Physicians, 21 S. 22nd St., Philadelphia.

The Pittsburgh Roentgen Society.—President, Zoe A. Johnston, M.D., 601 Jenkins Arcade; *Vice-president,* Prentiss A. Brown, M.D., and *Secretary-Treasurer,* Harold W. Jacox, M.D., 4800 Friendship Ave. Meetings held second Wednesday of each month at 4:30 P.M., from October to June at various hospitals designated by program committee.

RHODE ISLAND

See New England Roentgen Ray Society.

SOUTH CAROLINA

South Carolina X-ray Society.—President, Percy D. Hay, Jr., M.D., McLeod Infirmary, Florence; *Secretary-Treasurer,* Hillyer Rudisill, Jr., M.D., Roper Hospital, Charleston. Meetings in Charleston on first Thursday in November, also at time and place of South Carolina State Medical Association.

SOUTH DAKOTA

Meets with Minnesota Radiological Society.

TENNESSEE

Memphis Roentgen Club.—Chairmanship rotates monthly in alphabetical order. Meetings second Tuesday of each month at University Center.

Tennessee Radiological Society.—President, Steve W. Coley, M.D., Methodist Hospital, Memphis; *Vice-president,* Eugene Abercrombie, M.D., 305 Medical Arts Bldg., Knoxville; *Secretary-Treasurer,* Franklin B. Bogart, M.D., 311 Medical Bldg., Chattanooga. Meeting annually with State Medical Society in April.

TEXAS

Texas Radiological Society.—President, C. F. Crain, M.D., Corpus Christi; *President-elect,* M. H. Glover, M.D., Wichita Falls; *First Vice-president,* G. D. Carlson, M.D., Dallas; *Second Vice-president,* P. E. Wigby, M.D., Dallas; *Secretary-Treasurer,* L. W. Baird, M.D., Scott and White Hospital, Temple. Meets annually. The next annual meeting is to be Jan. 18, 1941, in Sherman.

VERMONT

See New England Roentgen Ray Society.

VIRGINIA

Radiological Society of Virginia.—President, Fred M. Hodges, M.D., 100 W. Franklin St., Richmond; *Vice-president,* L. F. Magruder, M.D., Raleigh and College Aves., Norfolk; *Secretary,* V. W. Archer, M.D., University of Virginia Hospital, Charlottesville.

WASHINGTON

Washington State Radiological Society.—President, H. E. Nichols, M.D., Stimson Bldg., Seattle; *Vice-president,* George Cornett, M.D., Yakima; *Secretary-Treasurer,* Kenneth J. Holtz, M.D., American Bank Bldg., Seattle. Meetings fourth Monday of each month at College Club, Seattle.

WISCONSIN

Milwaukee Roentgen Ray Society.—President, H. W. Hefke, M.D.; *Vice-president,* Frederick C. Christensen, M.D.; *Secretary-Treasurer,* Irving I. Cowan, M.D., Mount Sinai Hospital, Milwaukee. Meets monthly on first Friday at the University Club.

Radiological Section of the Wisconsin State Medical Society.—Secretary, Russel F. Wilson, M.D., Beloit Municipal Hospital, Beloit. Two-day annual meeting in May and one day in connection with annual meeting of State Medical Society, in September.

University of Wisconsin Radiological Conference.—Secretary, E. A. Pohle, M.D., 1300 University Ave., Madison, Wis. Meets every Thursday from 4 to 5 P.M., Room 301, Service Memorial Institute.

EDITORIAL

LEON J. MENVILLE, M.D., *Editor* HOWARD P. DOUB, M.D., *Associate Editor*

IS THE PRACTICE OF ROENTGENOLOGY ABOVE CRITICISM?

To one who has practised roentgenology for thirty-three years, it might be permissible to express some observations and, possibly, criticisms of the progress made in this specialty.

During the World War, in the need for roentgenologists, a large number were trained for this purpose. Considering the emergency at that time, they were well trained, but the instruction was insufficient for the private practice of this specialty, which many of these men took up on their return. The value of the x-ray was forcefully demonstrated at that time, with the result that when the War was over and doctors returned to civil practice, they demanded x-ray in their hospitals, even in the smaller towns. This created a demand for a larger number of roentgenologists. As the number of large hospitals (being more or less teaching centers for the universities) increased, another demand was made for more roentgenologists. The training of roentgenologists was begun in the medical courses and, later, special house-officer positions were created for those who intended to enter the specialty of roentgenology, with the result that, in a few years, a large number did enter.

Coming to more recent years, industries have begun to realize the value of medical care of workmen; again an increase in the use of the x-ray occurred. The establishment in various States of Workmen's Compensation Acts added to the need for roentgenology. Furthermore, in the last fifteen years there has been a marked increase in the number of diagnoses and cases of injury resulting from automobile accidents, all of which has increased the amount of work, particularly for the roentgenologist. As we have lived through these various phases of the building up of this specialty, with their marked increase in the use of the x-ray in all phases of civil life, we have been rather appalled at the ease with which diagnoses can be made from the x-ray and yet are so often found to be wrong.

One of the faults of the early German practice of roentgenology, as it came to us as observers, was that the roentgenologist was primarily a trained internist and his interpretations were 90 per cent clinical and 10 per cent x-ray. We do not believe this situation has existed in more recent years in this country, in spite of the gastro-enterologists and the various specialists installing their own equipment for their own purposes. We believe that these specialists who attempt the examination of their own patients by the x-ray cannot help but be biassed by the clinical findings, primarily, and, secondarily, by what the x-ray may show. Perhaps for this reason we are of the opinion that one of the most serious faults of the younger roentgenologist is that, when a case is referred to him, he feels he must make some sort of positive diagnosis in order to retain his consultant. When the case is negative, he is not willing to say so, apparently in fear of the consultant's opinion. Again, if he has had any degree of clinical experience, he is most likely to consider the clinical picture first and that of the x-ray second. One of the great values of the x-ray, it always seems to us, is the lack of bias on the part of the diagnostician, in spite of what the clinical diagnosis might have been or might prove to be. And thus it seems that a negative opinion, a report of a normal condition, or an inability to demonstrate the result of a trauma is as valuable to everyone concerned as a positive diagnosis. We have felt, however, that this was not the opinion of a very large group of roentgenologists.

In the private practice of roentgenology, with some exceptions, it makes little difference if, for example, the roentgenologist calls a change in a vertebra "hypertrophic arthritis" or "hypertrophic change," since the clinician will treat the patient on the basis of the clinical symptoms and not on that of the diagnosis. On the other hand, if the roentgenologist passes an opinion of a fracture, such as a fractured ankle,

and is perhaps wrong in his diagnosis, it makes little difference to the consultant because he will treat the case under the diagnosis and no harm will be done.

When we consider the more complicated compensation work, automobile liability, or accidents in the automotive industry, an accurate and correct diagnosis is of the utmost importance, because, eventually, either in the quasi-court of law or before the higher courts, such diagnoses are evaluated in damages in the form of dollars and cents. The physician who makes either a negative or positive diagnosis of a given condition will be called upon to prove this diagnosis. At the same time, roentgenologists, among others, are called upon to disprove positive diagnoses, and we must have facts which will substantiate our opinion as it differs from that of our colleagues. Mere opinions are not acceptable; one must offer fundamental as well as scientific facts in attempting to prove a positive or negative diagnosis. It is somewhat disturbing in this work to see so many x-ray films, from various parts of the country, submitted by large insurance companies, industries, and railroads, and to find diagnoses which have been made without any basis of fact whatsoever.

An example of this is the fact that, in twenty-one court appearances in one month, we differed eighteen times—practically a difference of 100 per cent—from the original diagnoses. This is rather startling to us because either those who made the positive diagnoses were wrong or we who made the negative diagnoses were wrong. In a careful analysis in a series of cases such as this and with the facts placed before an unbiassed committee of roentgenologists, doubtless there would be 100 per cent agreement on the negative against the positive, or, in this series, at least sixteen cases out of the eighteen. The difference in the other two cases would be academic, for example, the age of a fracture, if it were a fracture case. In analyzing these cases, which we try to do in every instance for our own information and instruction, there appears to be a startling lack of the fundamentals of diagnosis, with no thought of the many varied congenital anomalies of the whole skeleton. If the congenital anomalies are recognized, they are usually recognized as a cause of symptoms in most part, and, if not the cause of symptoms, the possibility of aggravation of symptoms. We believe that a general statement can be made, with few reservations, that none of the congenital anomalies, so commonly and frequently found, is the source of any weakness in structure or symptoms, either by themselves or with accident. Nor do we believe that owing to the defect symptoms are more likely to appear than in the absolutely normal individual.

From the plaintiff's side of the diagnosis, many medico-legal cases are based upon some minor variation. Space will not permit us to cite these, but I will use one, and that is spondylolisthesis. After many years of study, we have come to the firm conviction that, with rare exception, this condition is developmental in origin, possibly from fetal life. When trauma is the cause of this condition, it takes only a few weeks' study of the part to demonstrate definitely by proliferative and reactionary changes that this is the result of the alleged accident.

The second failure of correct interpretation would seem to be a lack of knowledge or interest in whether the change found in the given part is the result of the alleged accident or by its very appearance existed at the time of the accident. This reverts to a rather simple fundamental knowledge of time-element in the production of reactionary changes. Proliferative changes, healing or destructive areas, or calcifications appear and are diagnosed as the result of an alleged injury, in some instances, of from three to five weeks. Here again, when these changes are interpreted as the result of an alleged accident, one can say that there is a fundamental lack of knowledge of the pathologic changes which occur.

Again, when one hears in court the allegation that atrophy in the bones of the shoulder girdle in a well-nourished individual can be demonstrated in the x-ray film in from two days to one week, one feels that roentgenology has progressed rapidly in its ability to demonstrate changes which, to this writer, would seem impossible in this short period of time, much less visible by the x-ray film. In the last analysis, perhaps the x-ray cannot do all we had hoped for it or expected it would; nevertheless, it has its definite place in medicine, and its study is a science of exactness provided we—those of us who interpret—can be exact.

ARIAL W. GEORGE, M.D.

ANNOUNCEMENT

THE NEXT ANNUAL MEETING

The Twenty-sixth Annual Meeting of the Radiological Society of North America will be held at Cleveland, Ohio, in the Hotel Statler, on December 2, 3, 4, 5, and 6, 1940.

COMMUNICATIONS

DR. GEORGE E. PFAHLER HONORED

On Thursday evening, January 25, a testimonial dinner was given Dr. George E. Pfahler by the Philadelphia Roentgen Ray Society at The Warwick, Philadelphia.

Two hundred and fifty friends of Dr. Pfahler came from afar as well as from Philadelphia and its environs.

Dr. Eugene P. Pendergrass was Toastmaster. In the absence of Dr. Joseph E. Roberts, Jr., President of the Society, Dr. Jacob H. Vastine, II, Vice-president, read a message from Dr. Roberts. Other speakers were Dr. Francis F. Borzell, President-elect of the Medical Society of the State of Pennsylvania, Dr. George H. Meeker, Dean of the Graduate School of Medicine of the University of Pennsylvania, and Dr. Joseph McFarland, Professor of Pathology at the University of Pennsylvania. Dr. B. R. Kirklin, Secretary of the American Board of Radiology, read a tribute to Dr. Pfahler written by Dr. Percy Brown, Historian of the American Roentgen Ray Society.

As a memento of the occasion, Dr. W. Edward Chamberlain presented Dr. Pfahler with a silver platter from the Society. Dr. Pfahler accepted the platter with a gracious and modest speech.

The meeting was a pleasant one and this public acknowledgment of the debt which radiology owes Dr. Pfahler, and of the respect and affection in which his confrères and friends hold him, was evident from the large attendance and the remarks which were made.

JOHN T. FARRELL, JR., M.D.

RADIOLOGY wishes to join with those friends of Dr. Pfahler who were permitted to break bread with him, in congratulating him upon his remarkable record in radiology. The Philadelphia Roentgen Ray Society honored itself in honoring him. We like to think over the genial wit and wisdom which must have flowed about that banquet table, at which were gathered the leading exponents of our specialty.

The dinner was attended by Dr. Bernard H. Nichols, President of the Radiological Society of North America, and by Dr. Donald S. Childs, Secretary-Treasurer of that same Society, who expressed the felicitations of our members upon this happy occasion.

Not because of Dr. Pfahler's years of service in the teaching and practice of radiology do we delight to do him honor, but principally because of his ripe scholarship, the wealth of experience which is his, and his helpfulness toward those who have yet to attain their experience. What he has found out for himself he is ready to pass on to others.

RADIOLOGY adds its congratulations, Dr. Pfahler!

MID-WESTERN RADIOLOGISTS' FOURTH ANNUAL CLINICAL CONFERENCE

The Fourth Annual Clinical Conference of Mid-western Radiologists was held at the Brown Hotel, Louisville, Kentucky, on February 9 and 10, 1940. Presentations on many subjects of interest to radiologists were made by some of the leading physicians of Kentucky.

At the dinner meeting Dr. William Allen Pusey, of Chicago, spoke on the subject, "Reminiscences of My Early Experiences with the Roentgen Ray." He presented his office record and case history of the first patient he had ever treated with the roentgen ray, this being done early in the year 1900. He spoke of his therapeutic experiences with the roentgen ray in lesions of many types, many of which were undoubtedly so treated for the first time.

Dr. Irvin Abell, of Louisville, Kentucky, then spoke on the subject, "The Present United States Public Health Program." In prefacing his major presentation he paid a glowing tribute to the radiologist. He spoke of his great importance in the general field of medicine and of the many contributions to medical progress that had been made by radiologists during the last forty years. He also commended radiologists in general for the work that they were doing in preserving their specialty and for their defense against the attempted inroads of so-called Social Legislation. He then gave a remarkable summary of

the legislative activities of our Government in the field of medicine during the past six years, together with a summary of the legislation now before the Congress.

There was a Symposium on the Chest and a Symposium on Carcinoma of the Lung, each comprising five papers, as well as almost thirty papers on various radiologic subjects, presented by leading physicians.

Dr. C. A. Good, of the Mayo Clinic, in behalf of Dr. B. R. Kirklin, invited the Conference to meet in Rochester, Minnesota, next year. The invitation was accepted by the Conference.

THE AMERICAN BOARD OF RADIOLOGY

The next examination to be conducted by The American Board of Radiology will be in New York City, June 7–9, 1940, but the quota is practically filled for this examination. We would, therefore, urge those who are anxious to be examined this year to send their applications in immediately so that they may be scheduled for the examination to be conducted in Boston, Mass., Sept. 27–29, 1940.

Following is a list of those who have been certified or granted additional certification during 1939.

Address all communications to B. R. Kirklin, M.D., Secretary The American Board of Radiology, Mayo Clinic, Rochester, Minnesota.

The following were issued certificates in 1939:

	Name	Address	Field
1.	Abrams, Hyman S.	Tuscaloosa, Ala.	Radiology
2.	Ackermann, Alfred J.	Oklahoma City, Okla.	Roentgenology
3.	Algin, Sergius	Indiana, Pa.	Roentgenology
4.	Allen, William E., Jr.	St. Louis, Mo.	Radiology
5.	Almy, Max A.	Rochester, N. Y.	Roentgenology
6.	Andrew, F. D.	Rochester, N. Y.	Radiology
7.	Ansprenger, Aloys G.	Macomb, Ill.	Radiology
8.	Athle, Laxman H.	Bombay, India	Radiology
9.	Bachman, Arnold L.	Stamford, Conn.	Radiology
10.	Baltimore, Louis	Los Angeles, Cal.	Roentgenology
11.	Barner, John L.	Cincinnati, O.	Therapeutic Radiology
12.	Bayliss, Jacob W.	Buffalo, N. Y.	Roentgenology
13.	Beatty, Samuel R.	Denver, Colo.	Radiology
14.	Bell, Charles E.	East St. Louis, Ill.	Radiology
15.	Bernstein, Alfred J.	New York, N. Y.	Radiology
16	Bersack, Solomon R.	Springfield, Mass.	Radiology
17.	Bond, Thomas B.	Fort Worth, Texas	Roentgenology
18.	Bonis, Alexander	New York, N. Y.	Radiology
19.	Boswell, Frederick P.	Montgomery, Ala.	Radiology
20.	Bracher, George J.	Seattle, Wash.	Diagnostic Roentgenology
21.	Brindle, Harry R.	Asbury Park, N. J.	Roentgenology
22.	Brown, William L., Sr.	Chicago, Ill.	Radium Therapy
23.	Bryan, William W.	Montreal, Canada	Radiology
24.	Bugbee, Edwin P.	Philadelphia, Pa.	Radiology
25.	Burvill-Holmes, E.	Philadelphia, Pa.	Radiology
26.	Butler, William J.	Providence, R. I.	Radiology
27.	Carr, Gladys L.	Hempstead, N. Y.	Roentgenology
28.	Carroll, William J.	Hot Springs, Ark.	Radiology
29.	Casper, Stephen L.	Philadelphia, Pa.	Radiology
30.	Caulk, Ralph M.	Washington, D. C.	Radiology
31.	Chang, C. William	New York, N. Y.	Radiology
32.	Cohen, Paul W.	New York, N. Y.	Therapeutic Radiology
33.	Colton, Benjamin	Flushing, N. Y.	Diagnostic Roentgenology
34.	Conahan, Thomas J.	Hazleton, Pa.	Radiology
35.	Cooney, James P.	Washington, D. C.	Radiology
36.	Cooper, George, Jr.	University, Va	Roentgenology
37.	Cooper, Robert W.	Shreveport, La.	Radiology
38.	Curtzwiler, Francis C.	Toledo, O.	Radiology
39.	Daversa, Joseph J.	Brooklyn, N. Y.	Roentgenology
40.	Dell, J. Maxey	Gainesville, Fla.	Roentgenology
41.	Doble, Eugene H.	Presque Isle, Me.	Diagnostic Roentgenology

42.	Dowdy, Andrew H.	Rochester, N. Y.	Radiology
43.	Echternacht, Arthur P.	Indianapolis, Ind.	Radiology
44.	Elliott, William J.	Worcester, Mass.	Radiology
45.	Epstein, Bernard S.	Brooklyn, N. Y.	Radiology
46.	Euphrat, Edwin J.	Elkins, West Va.	Radiology
47.	Evans, William A., Jr.	Detroit, Mich.	Radiology
48.	Fierstein, Jacob	New York, N. Y.	Roentgenology
49.	Flanagan, E. Latane	Richmond, Va.	Roentgenology
50.	Gartenlaub, Charles	New York, N. Y.	Roentgenology
51.	Gilbert, Philip D.	Camden, N. J.	Radiology
52.	Gilmore, John H.	Chicago, Ill.	Radiology
53.	Good, C. Allen	Rochester, Minn.	Radiology
54.	Grady, Mary Alice	San Francisco, Cal.	Roentgenology
55.	Gray, Charles M.	Tampa, Fla.	Radiology
56.	Grimm, Homer W.	Pittsburgh, Pa.	Radiology
57.	Hall, James B.	Johnstown, Pa.	Roentgenology
58.	Hankins, Walter D.	Johnson City, Tenn.	Roentgenology
59.	Hanley, D. Reed	Streator, Ill.	Roentgenology
60.	Hartung, Walter	Iowa City, Ia.	Radiology
61.	Harwell, Wilbur R.	Shreveport, La.	Radiology
62.	Hayes, Arthur W.	Greenfield, Mass.	Diagnostic Roentgenology
63.	Herrmann, Henry C.	Louisville, Ky.	Therapeutic Radiology
64.	Hiebert, Peter E.	Kansas City, Kans.	Radiology
65.	Hill, John M.	Pittsburgh, Pa.	Radiology
66.	Hilt, Lawrence M.	Grand Rapids, Mich.	Radiology
67.	Hobbs, Arthur A., Jr.	Philadelphia, Pa.	Radiology
68.	Holtz, Harvey E.	Dallas, Tex.	Radiology
69.	Howard, Corbett E.	Goldsboro, N. C.	Therapeutic Radiology
70.	Howe, Martha E.	New York, N. Y.	Therapeutic Radiology
71.	Hummon, Irvin F., Jr.	Oak Park, Ill.	Roentgenology
72.	Hunter, Arthur F.	New York, N. Y.	Roentgenology
73.	Jaffrey, George	Olean, N. Y.	Radiology
74.	Jelte, Safford A.	Oakland, Cal.	Radiology
75.	Jenkins, I. Warner	Waco, Tex.	Radiology
76.	Jenkins, William F.	Columbus, Ga.	Roentgenology
77.	Jensen, Herman H.	Minneapolis, Minn.	Radiology
78.	Jimenez, Juan M. M.	New York, N. Y.	Radiology
79.	Johnson, Roy W.	Los Angeles, Cal.	Radiology
80.	Johnston, Wayne A.	Dubuque, Ia.	Roentgenology
81.	Kelley, Charles H.	Washington, D. C.	Radiology
82.	Kellogg, Douglas S.	Ft. Sam Houston, Tex.	Roentgenology
83.	Kestel, John L.	Waterloo, Ia.	Diagnostic Roentgenology
84.	Kirsh, Israel E.	Salt Lake City, Utah	Radiology
85.	Koenig, Edward C.	Buffalo, N. Y.	Roentgenology
86.	Lake, William F.	Atlanta, Ga.	Roentgenology
87.	Landham, Jackson W.	Atlanta, Ga.	Roentgenology
88.	Larkin, John C., Jr.	Berkeley, Cal.	Radiology
89.	Lavine, Sidney B.	Trenton, N. J.	Diagnostic Roentgenology
90.	Lavner, Gerald	Haverhill, Mass.	Diagnostic Roentgenology
91.	Lawlah, John W.	Chicago, Ill.	Radiology
92.	LeWald, Leon T.	New York, N. Y.	Radiology
93.	Leader, Samuel A.	Lexington, Ky.	Diagnostic Roentgenology
94.	Lerner, Henry H.	Boston, Mass.	Diagnostic Roentgenology
95.	Levy, Abraham H.	Brooklyn, N. Y.	Radiology
96.	Lewis, Elbert K.	Chicago, Ill.	Radiology
97.	Lia, Bianca Rachel	Boston, Mass.	Roentgenology
98.	Liberson, Frank	Brooklyn, N. Y.	Roentgenology
99.	Logan, George E. C.	Philadelphia, Pa.	Radiology
100.	McAlister, Lawrence S.	Muskogee, Okla.	Radiology
101.	McCampbell, Herbert H.	Knoxville, Tenn.	Radiology
102.	McCandless, Oliver H.	Kansas City, Mo.	Roentgenology
103.	McCullough, Thomas L.	Pittsburgh, Pa.	Diagnostic Roentgenology
104.	McGreer, John T.	Dayton, O.	Radiology
105.	McKinney, G. C.	Lake Charles, La.	Roentgenology
106.	McNattin, Robert F.	Chicago, Ill.	Therapeutic Radiology
107.	Mackoy, Frank W.	Milwaukee, Wis.	Diagnostic Roentgenology
108.	Magruder, Levin W.	New Orleans, La.	Radiology
109.	Manley, Louis V.	Northampton, Mass.	Diagnostic Roentgenology
110.	Martineau, Lawrence A.	Providence, R. I.	Roentgenology
111.	Mass, Max	Macon, Ga.	Diagnostic Roentgenology
112.	Maxfield, James R., Jr.	Waco, Texas	Radiology
113.	Meader, Robert P.	Philadelphia, Pa.	Radiology
114.	Mokrohisky, Stephen M.	Green Bay, Wis.	Roentgenology
115.	Moore, J. N.	Ocala, Fla.	Roentgenology
116.	Morgan, Arthur E.	Washington, Pa.	Roentgenology
117.	Morgan, Harold W.	Mason City, Ia.	Radiology

118.	Morris, Clyde L.	Peoria, Ill.	Roentgenology
119.	Myers, Karl J.	Philippi, West Va.	Radiology
120.	Nash, Newman C.	Wichita, Kans.	Radiology
121.	Neely, John M.	Lincoln, Neb.	Radiology
122.	Newton, John O.	Cleveland, O.	Roentgenology
123.	Norton, Richard C.	Battle Creek, Mich.	Diagnostic Roentgenology
124.	Palmer, James P.	Bronx, N. Y.	Radiology
125.	Payne, Andrew K.	Jackson, Mich.	Radiology
126.	Pearlstein, Frank	West New York, N. J.	Diagnostic Roentgenology
127.	Pedersen, Nicholas S.	San Francisco, Cal.	Roentgenology
128.	Phillips, Allan B.	Des Moines, Ia.	Radiology
129.	Piatt, Arnold B.	Tulsa, Okla.	Roentgenology
130.	Plaut, H. F.	Mansfield, O.	Radiology
131.	Pollack, Simon	St. Louis, Mo.	Roentgenology
132.	Popma, Alfred M.	Boise, Idaho	Radiology
133.	Poppel, Maxwell H.	New York, N. Y.	Roentgenology
134.	Prince, Norman C.	Amarillo, Tex.	Roentgenology
135.	Reed, Harold R.	Washington, D. C.	Radiology
136.	Reid, Edward K.	Rome, N. Y.	Diagnostic Roentgenology
137.	Rhinehart, B. A.	Little Rock, Ark.	Roentgenology
138.	Richardson, Maurice L.	Freeport, Ill.	Roentgenology
139.	Roberto, Romeo	Yonkers, N. Y.	Diagnostic Roentgenology
140.	Roberts, Edward W.	Chicago, Ill.	Roentgenology
141.	Robilotti, James G.	New York, N. Y.	Diagnostic Roentgenology
142.	Rodes, Charles B.	Butte, Mont.	Diagnostic Roentgenology
143.	Rogers, Frank T.	Ann Arbor, Mich.	Radiology
144.	Rubin, J. S.	Jamaica, N. Y.	Diagnostic Roentgenology
145.	Sackett, George L.	Cleveland, O.	Radiology
146.	Scott, Harry A.	Lincoln, Neb.	Diagnostic Roentgenology
147.	Senturia, Hyman R.	Chicago, Ill.	Radiology
148.	Sherman, Cyril F.	Wichita, Kans.	Roentgenology
149.	Sickel, Emanuel M.	Lakewood, N. J.	Diagnostic Roentgenology
150.	Silverstone, Sidney M.	New York, N. Y.	Therapeutic Radiology
151.	Singer, Bella	Elizabeth, N. J.	Diagnostic Roentgenology
152.	Sirca, Dionisie M.	Springfield, Ill.	Roentgenology
153.	Sisk, J. Newton	Madison, Wis.	Radiology
154.	Smith, Barney B.	Buffalo, N. Y.	Roentgenology
155.	Smith, Earl D.	Utica, N. Y.	Radiology
156.	Smith, Ernest E.	New York, N. Y.	Radiology
157.	Sokow, Theodore	Kenosha, Wis.	Radiology
158.	Solomon, Bennet	Springfield, Mass.	Diagnostic Roentgenology
159.	Southworth, J. D.	Rutland, Vt.	Therapeutic Radiology
160.	Spencer, Earle W.	Saskatoon, Sask.	Radiology
161.	Stacy, Archie J.	Tupelo, Miss.	Diagnostic Roentgenology
162.	Sternbergh, W. C. A.	Clifton Springs, N. Y.	Radiology
163.	Stewart, Calvin B.	Atlanta, Ga.	Therapeutic Radiology
164.	Szymanski, John J.	Passaic, N. J.	Radiology
165.	Talley, Louis F.	Marshalltown, Ia.	Roentgenology
166.	Thompson, William G.	Holden, Mo.	Radiology
167.	Truog, Clarence P.	Minneapolis, Minn.	Roentgenology
168.	Tyner, Furman H.	Port Arthur, Tex.	Radiology
169.	Vaughn, Robert J.	Annapolis, Md.	Roentgenology
170.	Wallace, William S.	Durham, N. C.	Radiology
171.	Ward, Leo J.	Elizabeth, N. J.	Diagnostic Roentgenology
172.	Warden, Ralph H.	Chicago, Ill.	Roentgenology
173.	Webb, Harold H.	Ottumwa, Ia.	Radiology
174.	Wehr, William H.	Buffalo, N. Y.	Therapeutic Radiology
175.	Weinstein, Samuel	Brooklyn, N. Y.	Roentgenology
176.	Weitz, Harry L.	Traverse City, Mich.	Roentgenology
177.	Williams, Edwin G. C.	Danville, Ill.	Therapeutic Radiology
178.	Willis, Augusta Elizabeth	Orangeburg, S. C.	Roentgenology
179.	Wilson, Angus K.	Norfolk, Va.	Radiology
180.	Wilson, Russell F.	Beloit, Wis.	Radiology
181.	Worth, Harry M.	Victoria, B. C.	Radiology
182.	Wright, Hobart H.	Ann Arbor, Mich.	Radiology
183.	Zielinski, John B.	Fall River, Mass.	Radiology

A NOTE OF CORRECTION

Word has been received from James F. Brailsford, M.D., of Birmingham, England, that the word "chondroma" (Radiology, 33, 494, October, 1939), fourth line from bottom of page, first column, and in the caption to Figure 10 on the same page, should read "chordoma."

IN MEMORIAM

Word has been received of the death of four members of the Radiological Society of North America: Milo Wilson, M.D., of Gallipolis, Ohio; Prof. F. K. Richtmyer (associate), of Ithaca, N. Y.; Morgan D. Baker, M.D., of San Jose, Calif., and Bernhard Friedlaender, M.D., of Detroit, Michigan. The friends and associates of these late members will grieve to learn of their passing.

BOOK REVIEW

DIFFERENTIALDIAGNOSTIK DER LUNGENERKRANKUNGEN IM RÖNTGENBILDE (Roentgenographic Differential Diagnosis of Diseases of the Lungs), with Special Consideration of the Lateral Position. By Dr. O. SIMON, head of the Roentgenologic Division of the Medical Clinic, University of Koenigsberg. A volume of 84 pages, with 204 illustrations. Published by Georg Thieme, Leipzig, 1939. Price: 19.00 R.M. (25 per cent discount allowed to foreign purchasers).

This small volume, another one of the series of supplements to the *Fortschritte auf dem Gebiete der Röntgenstrahlen*, is essentially an atlas, the major portion being occupied by excellent positive reproductions of postero-anterior and lateral roentgenograms of the chest. In the foreword by Professor Assmann, great stress is laid upon the need for films of the chest to be made in two positions, at right-angles to each other. Accordingly, the lateral views are emphasized.

The cases are grouped in unique fashion, various pathologic entities which produce similar roentgenologic findings being treated in each section. The discussion is divided into the following groups: extra-pulmonary involvement including chest wall processes and pleural lesions; small sized pulmonary lesions, including miliary processes, vascular changes, etc., involvement of gross pulmonary segments, such as atelectasis, pneumonia, tuberculosis, etc., cavitations and enlargements of lymph nodes; changes in the diaphragms, and finally, bronchial tumors. The illustrations are well selected and instructively grouped. This is an excellent volume for the study of the differential diagnosis of pulmonary and pleural diseases.

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JOSEPH T. DANZER, M.D., of Oil City, Penna.
SYDNEY J. HAWLEY, M.D., of Danville, Penna.
HANS W. HEFKE, M.D., Milwaukee, Wisc.
JOHN B. MCANENY, M.D., of Madison, Wisc.
ANTONIO MAYORAL, M.D., of New Orleans, La.
JOHN M. MILES, M.D., of Lafayette, La.
LESTER W. PAUL, M.D., of Madison, Wisc.
HAROLD O. PETERSON, M.D., of Minneapolis, Minn.
ERNST A. POHLE, M.D., Ph.D., of Madison, Wisc.
ERNST A. SCHMIDT, M.D., of Denver, Colo.
CHARLES G. SUTHERLAND, M.D., of Rochester, Minn.
WILLIS A. WARD, M.D., of Chicago, Ill.

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paratus which the author describes. In examining the mastoid, an exposure 8 cm. in diameter is made. This film is used as a guide, with the patient's head well immobilized, and smaller diametered exposures are made of various parts of the mastoid. The use of the small fields gives a sharp image. Breaking the examination down into small units enables the examiner to equalize better the difference in density of the various parts. By the use of varying incidences of the rays in different parts of the mastoid, more accurate interpretation is possible. The method is useful also in exploring various parts of the cranium.

Tomography.—The author uses repeated exposures on the same film, moving the tube and film as in tomography, but during the exposure the tube and film are motionless. The linear images produced by the parts not in focus are represented by a series of small dots. As they are clearer than the blurred images in ordinary tomography of the structures not in the focal plane, some information about them can be obtained. The stereographic centering apparatus described may be used for this purpose. It will also indicate the level of the focal plane. The technic for using the apparatus in this manner is described.

PART II

By adding lights to the stereoradiographic apparatus, a device is obtained which readily localizes opaque foreign bodies in the body and which can be used to guide the surgeon in finding them. The technic for operating the device is given in detail.

SYDNEY J. HAWLEY, M.D.

BIOLOGIC EFFECTS

Functional Factors in Radiation Therapy. M. Bertolotti. Strahlentherapie, **65**, 87, 1939.

The author analyzes a number of problems touching the relationship between physically absorbed dose and biologic effect. He contends that in addition to the purely physical explanation that the biologic effect of radiation on tissues is a direct function of the absorbed radiant energy, a physiologic factor must be considered which takes into account the properties of the irradiated tissues as well as the place of absorption. The rôle of the sympathetic nervous system in this connection is also discussed.

ERNST A. POHLE, M.D., Ph.D.

Experimental Studies and Theoretical Deductions Regarding the Hypothesis of the Biologic Effect of Roentgen and Gamma Rays on Living Matter. G. G. Palmieri and A. Valenti. Strahlentherapie, **65**, 15, 1939.

The authors studied the relationship between the absorption of radiant energy and the biologic reaction, using the skin erythema as test. If the two processes were running parallel, then the effect in the skin should decrease with increasing frequency of the radiation. However, it was found that for 2 kv. a surface dose of 6,000 r corresponded to the erythema dose while the corresponding figures for 150 and 500 kv. were 450 and 1,200 r, respectively. Consequently, the authors deduced that the effect on the skin is not proportional to the energy absorbed as measured by ionization. The possibility of a non-specific action or a catalytic effect in explanation of these observations is discussed.

ERNST A. POHLE, M.D., Ph.D.

BIOLOGIC EXPERIMENTATION

Further Studies with Seedlings on the Question of Wave Length Dependence of Radiobiological Reactions. P. S. Henshaw and D. S. Francis. Am. Jour. Cancer, **35**, 386–400, March, 1939.

Triticum and *Lycopersicum* seedlings were treated with 200 and 700 kv. x-rays. In some experiments the seedlings were surrounded with a low atomic weight material. *Lycopersicum* seedlings were more sensitive to 700 kv. radiation in all the experiments. They were more sensitive by 42 per cent when the low atomic material was used and by 122 per cent when it was not. These findings are taken as evidence that there is a differential response on the part of the organism to different qualities of radiation.

HAROLD O. PETERSON, M.D.

Some Unsolved Problems of Radiation Biology. P. del Buono. Strahlentherapie, **65**, 108, 1939.

The author undertook a series of experiments in order to study the effect of the wave length on the biologic effect. He exposed the seeds of peas, wheat, and millet to x-rays (from 140 to 180 kv., doses from 25 to 1,000 r, one, three, and six days after germination had begun). The length of the seedlings was carefully recorded and the results plotted in graphs. It appeared that for the type of radiation used and for any type of radiation there exists the possibility of stimulation. After any irradiation, even after a dose of 25 r, an injury even if slight must be expected. For doses of 100 r and above, the degree of injury increases with the dose. For plants there is a certain period during the growth when the radiosensitivity is at a maximum. It cannot be determined yet whether this is specific for the individual type of plant or whether it is due to a general biophysical factor. The phenomena observed were independent of the penetration of radiation and of the duration of the exposure. Consequently it is assumed that the biologic effect of radiation depends on the dose and the state of the organism at the time of irradiation.

ERNST A. POHLE, M.D., Ph.D.

BONE DISEASES (DIAGNOSIS)

Hereditary Deforming Chondrodysplasia: Report of Ten Cases in One Family. Charles M. Graney. Jour. Am. Med. Assn., **112**, 2026–2030, May 20, 1939.

This condition was first described in 1825, and so concisely that the description embraces all that is of import regarding it. It has been perhaps best defined as "an hereditary disturbance of bone growth beginning early in childhood and characterized by multiple carti-

laginous and osteocartilaginous growths within or on the skeletal system." Malignant degeneration is said to occur in 5 per cent of cases, but the incidence is probably less than that. In diagnosis, the hereditary factor, the symmetrical distribution of the exostoses, their multiplicity, and the x-ray appearances are important. X-ray examination shows multiple exostoses of the long bones occurring in the epiphyseal region but springing from the diaphysial side and directed away from the epiphyses along the line of muscular pull. The exostoses vary from simple spikes to pedunculated and cauliflower types and from well ossified spurs to typical chondromas. The epiphyses may be misshapen or absent, although they do not produce any exostoses themselves. The bones most frequently affected occur in this order: ulna, fibula, femur, humerus, and phalanges. The carpus, tarsus, vertebræ, skull, and sternum are not involved, since pure cartilaginous or membranous bone escapes this process.

Ehrenfried, in 1915 and 1917, found that the reported cases in the literature totalled about 700.

The etiology of this condition is obscure. The neoplastic theory is not tenable, as growth and development of exostoses stop at the age of from 20 to 22 years with the attainment of skeletal maturity. It is apparently unrelated to rickets or vitamin deficiency; if it were so related, there should be no hereditary factor. The distribution of the disease is the same as that of a thyroid deficiency, and the possibility of a glandular disturbance, as of thyroid or pituitary, has been considered. A failure of the process of breaking down of bone and shaping or modeling of the bone, as described by John Hunter, has been suggested as the cause. Another theory is that it is produced by impressions made on the germ plasm, consequent to fractured bones and their complex healing processes or that the patients "are made susceptible through transmission from past generations of various obnoxious environmental experiences."

These conditions should remain untreated until after bony growth has stopped. Indications for removal are deformity, interference with the function of joints, muscles, or tendons, and evidence of a malignant condition or an increase in size after bony growth has stopped.

CHARLES G. SUTHERLAND, M.D.

The Endocrine Implication of Juvenile Chondro-epiphysitis. Robert L. Schaefer, Fred L. Strickroot, and Frank H. Purcell. Jour. Am. Med. Assn., **112**, 1917–1919, May 13, 1939.

Juvenile chondro-epiphysitis is described as an alteration in shape of the developing epiphyses associated with an apparent aseptic necrosis. Pathologically there may be rarefaction or even cyst-like formation and degeneration. Clinically there is possibility of eventual deformity. Roentgenographically it is characterized by the following changes in the developing osseous centers: diminution in size, decrease in density, rarefaction, fragmentation, irregularity of the ossification contour, and, in general, a fuzzy, ragged appearance of the chondro-epiphyseal structures. Depending on the various structures showing such x-ray changes, almost innumerable proper or descriptive names have been employed. This conglomeration of proper names is confusing in conditions which, we think, are identical and have the same etiology.

The significance of this publication is an attempt to prove that juvenile chondro-epiphysitis is due to an endocrine imbalance, specifically to a primary or secondary hypothyroidism in which the tissue-differentiating factor is absent, that it is a generalized process involving one or more of the developing chondro-epiphyseal structures, and that it is, at least, incipiently asymtomatic.

The roentgenographic study of osseous development ranks to-day as one of our most useful diagnostic procedures. In the age group from birth to eight years it by far outranks the basal metabolic rate for accuracy. In the older age group, from juvenility to completion of adolescence, delayed union or advanced union of the epiphyses gives visual evidence in the x-ray of premature adolescence or sex maturity or of gonadal hypofunction.

In 1931, the authors began to do routine x-ray examinations of all children with suspected endocrinopathies. Very early in their studies of endocrine dwarfism they were struck by the frequency of single or multiple involvement of the chondro-epiphyseal structures. It soon became apparent that this condition was not peculiar to dwarfism but was being seen with as great or greater frequency in other endocrine conditions displaying signs of hypothyroidism. Of 258 endocrinopathic patients studied, 91 (35.2 per cent) showed a chondro-epiphyseal disturbance. The number of hypothyroid cases showing chondro-epiphyseal disturbance was 85 (39.0 per cent). All but six cases were associated with hypothyroidism. Thirty-eight (41.7 per cent) of the 91 showing involvement had multiple epiphyseal lesions. The trochlea of the humerus was most frequently involved. There were only three patients in the entire group who had any symptoms referable to the osseous involvement. In a control group of 99 children with serial roentgenographic examinations at yearly intervals (totalling 2,200 films), only seven (7 per cent) showed evidence of chondro-epiphysitis.

The writers feel that x-ray evidence of this lesion is a definite indication for thyroid therapy.

CHARLES G. SUTHERLAND, M.D.

Punctate Epiphyseal Dysplasia Occurring in Two Members of the Same Family. D. G. Maitland. British Jour. Radiol., 12, 91–93, February 1939.

A case is reported of a female child, aged three days. The limbs were spastic, and the knee and elbow joints enlarged. Radiographic examination showed punctate areas of calcification in the spine, sacrum, and pelvis. The femora and humeri were shorter and thicker than normal; the proximal ends of the diaphyses were

widened, cupped, and irregular and showed punctate calcification. The mother was normal; the father had developmental defects of the fingers. One sister of the child reported had the same defects; two other siblings were normal.

SYDNEY J. HAWLEY, M.D.

BONE TUMORS

Metastatic Bone Carcinomatosis Associated with Parathyroid Hyperplasia and with Symptoms of Hyperparathyroidism: With Blood Calcium and Phosphorus Studies before and after Extirpation of a Hyperplastic Parathyroid Gland. S. Ben-Asher. Jour. Lab. and Clin. Med., 24, 709–717, April, 1939.

The author presents a case with metastatic carcinoma associated with parathyroid hyperplasia and with symptoms of hyperparathyroidism, such as hypercalcemia, a negative calcium balance, and increased excretion of calcium in the urine. The gland was removed and the blood calcium became lower, but not to the normal level.

This case and its discussion demonstrate the importance of the roentgenologist's consideration of the possibility of a co-existing hyperparathyroid disease when making a diagnosis of metastatic bone carcinomatosis.

This causes one to consider the hyperplasia of the gland as being secondary to the bone changes.

WILLIS A. WARD, M.D.

Tumors of Bone. Channing C. Simmons. New England Jour. Med., 220, 629–632, April 13, 1939.

The author adheres to the classification of bone tumors suggested by the Registry of Bone Sarcoma.

Non-malignant osteogenic tumors are usually readily diagnosed but one that contains a preponderance of cartilage should be removed for fear of malignancy.

Osteogenic sarcoma may be osteoblastic or osteolytic, and is described by the prefixes osteo-, chondro-, and myxo-. Metastasis is frequently early and the prognosis poor, no matter what the treatment. The x-ray film always shows bone destruction and bone formation. The periosteum may be elevated by ray formation and the so-called "reactive triangle" may be seen. In diagnosis, the history, film findings, and biopsy should be taken together.

Ewing's sarcoma arises from the shaft of long bones and is characterized by bone destruction with no tumor-bone formation although there may be some reactive new bone. Metastases to lungs and skull occur. Onion-skin appearance of the periosteum is characteristic. It may be confused with osteomyelitis xanthoma and reticulum-cell sarcoma. It is radiosensitive but metastases occur.

Reticulum-cell sarcoma is believed to arise from the reticulo-endothelial system and belongs to the lymphomas. An early x-ray film shows central bone destruction. The tumor is radiosensitive but the best results are believed to follow surgery.

Liposarcoma behaves like an osteogenic sarcoma.

Myeloma is a tumor of the marrow composed of plasma cells, usually multiple but possibly single. X-ray films show multiple areas of destruction. It is usually radioresistant.

The giant-cell tumor arises in the medulla near a joint, causing destruction and expansion of the cortex. The cavity is divided by fine trabeculæ and the tumor is sharply demarcated by a concavo-convex line. The tumor should be irradiated except in cases in which it can be easily removed surgically.

Central angioma of bone is rare and arises near the epiphyseal ends of long bones. It may resemble a giant-cell tumor but the trabeculæ are more marked. Treatment is the same as for the giant-cell tumor.

Metastatic tumors are variable.

Parosteal fibrosarcoma arises from the periosteum, involving bone secondarily and it is less malignant than true osteogenic sarcoma.

J. B. MCANENY, M.D

THE BREAST

The Clinical Significance of a Lump in the Breast. George Crile, Jr. Cleveland Clin. Quart., 6, 231–240, July, 1939.

Two hundred consecutive cases of breast tumors have been studied and 43 per cent were found to be malignant. Seventy-five per cent of these patients were over 50 years of age. Twenty per cent of the malignancies of the group occurred in the 40- to 50-year group. The only valuable clinical finding is dimpling of the skin, and that is not reliable. The discovery of axillary glands is a late finding. Diagnosis is best established by biopsy.

J. B. MCANENY, M.D.

Irradiation in the Treatment of Cancer of the Breast. U. V. Portmann. Cleveland Clin. Quart., 6, 109–115, April, 1939.

The greatest event in the treatment of cancer of the breast was the development of a rational surgical technic by Halsted. The percentage of five-year cures reported in the literature varies from 15 to 50 per cent, depending greatly on the choice of cases for operation; the average is about 30 per cent. Any means of increasing the number of cures should certainly be utilized. Classification of carcinoma of the breast based on microscopic examination has virtue but does not tell the whole story, omitting the extent of involvement, for one thing.

Portmann suggests a clinical grouping consisting of: Group I, which includes the freely movable localized tumor without metastasis or skin involvement; Group II, consisting of a freely movable localized tumor without skin involvement but with metastasis to the axillary nodes or other tissues, and Group III, in which there is diffuse involvement of the breast, skin involvement, edema, ulceration or nodules, and metastasis.

With this grouping in mind, 373 cases of breast cancer operated upon by Dr. George Crile were examined.

All cases of Group I lived five years; about 50 per cent of Group II cases lived five years, but in Group III no patient survived the five-year period. None of these patients received irradiation therapy.

Using the same standards of comparison, 235 cases that received post-operative irradiation were examined and it was found that irradiation was of no benefit in Group I cases because they all lived five years without irradiation. Among Group II cases, about 70 per cent survived the five-year period. The cases of Group III showed a five-year survival rate of 10 per cent, but none was without evidence of cancer. In addition, the proportion of patients who survived each yearly period up to five years was higher than the number of those who were operated upon only. What is more this study does not take into account the benefit derived from irradiation in metastasis.

The question of sterilization by pelvic irradiation is not discussed.

An important point presented by this author is a discussion of evidences of incurability, which is difficult to find in the literature. These evidences of incurability are grouped as edema, ulceration, nodules, induration, and inflammation of the skin; edema, diffuse infiltration, multiple tumors, and fixation of the tumor of the breast, and lymph node or distant metastasis.

J. B. McAneny, M.D.

CANCER (THERAPY)

Primary Squamous-cell Carcinoma of the Uvula: Report of Two Cases. J. Milton Robb and Merrill W. Michels. Ann. Otol., Rhinol., and Laryngol., 48, 521–524, June, 1939.

Primary squamous-cell carcinomas of the uvula are rare. The authors report two cases. Early excision offers a better prognosis than radiation alone, and this was done in the two cases reported. In one, external supervoltage (500 kv.) roentgen therapy was given post-operatively. Both patients have remained well.

Lester W. Paul, M.D.

Metastatic Cancer in the Lymph Nodes of the Neck. Harold G. F. Edwards. Southern Med. Jour., 32, 905–910, September, 1939.

Radiosensitivity is less in metastatic cancer of the lymph nodes of the neck than in the primary lesion, due, among other reasons, to inflammation, fibrosis, and degenerative changes in the nodes. Frequent examinations for involved nodes are necessary because of the latent period of from eight to ten months before metastasis reveals itself. When the nodes are involved, aspiration biopsy is first done, oral sepsis, if present, is reduced, and treatment instituted at once or a few days after completion of irradiation of the primary lesion.

Small nodes from a primary epidermoid cancer are treated with radium implants sufficient to deliver from seven to ten erythema doses. Small nodes from a transitional or lympho-epithelioma can sometimes be treated with x-rays in fractioned doses, giving 7,000 or 8,000 r over a field not exceeding 4 or 5 cm. Large areas of involvement with invasion of surrounding tissue are treated with external irradiation (200 r daily) for a total up to 3,500 r, followed immediately by transcutaneous implantation of radium needles to bring the total dosage up to from seven to ten erythema doses.

John M. Miles, M.D.

Treatment of Carcinoma of the Vallecula. E. Wessely. Strahlentherapie, 65, 601, 1939.

Since 1932, the author has treated 21 cases of cancer of the epiglottis and the base of the tongue. Nineteen were men, two were women; the age fluctuated between 50 and 60 years, the youngest being 38 and the oldest 73. Histologically, the tumors were mostly squamous-cell carcinoma; in over half of the cases there were definite glandular metastases. Most of the patients were given x-ray therapy applied according to the method of Coutard; the dose effective in the tumor was 2,500 r and given through two fields. Residual tumor tissue was treated with radium either by means of surface applicators or by interstitial irradiation (5 mg. needles). Five cases remained free from recurrence for over one year; one for two years; three for three years; one for four years; one for seven years, and one for eight years. In conclusion, the author emphasizes the importance of a thorough examination of the base of the tongue and the epiglottis in older persons who complain of vague disturbances when swallowing.

Ernst A. Pohle, M.D., Ph.D.

Radio-surgical Treatment of Carcinoma of the Stomach. A. Salotti. Strahlentherapie, 65, 121, 1939.

The author relates his experience in the treatment of carcinoma of the stomach by means of interstitial and contact radium therapy. After exposure of the tumor, from 1,176 to 2,400 mg.-hr. are applied interstitially or from 2,000 to 3,000 mg.-hr. if contact screens are used. This requires a treatment time of from 40 to 70 hours. No immediate or late injuries of the neighboring organs have been observed. A series of roentgenograms shows the needles *in situ* and also the roentgenologic appearance of the stomachs of some patients before and after treatment. The advantages of the method are that the stomach remains intact in the body, that it requires only an ordinary incision, and that it is apparently possible to give doses sufficiently high to destroy the tumor without injury to the surrounding normal tissue.

Ernst A. Pohle, M.D., Ph.D.

What can One Expect from Radiation in Carcinoma of the Rectum and Anus? Orville N. Meland. California and West. Med., 50, 403–407. June, 1939.

The author's discussion and review of the effects of radiation in carcinoma of the rectum and anus are very good. He divides his cases into three groups—inoperable, recurrent, and operable.

In the first group he hoped for nothing but palliation:

in the second group therapy was given to retard the growth, as well as to relieve pain. The patients in these first two groups had only a short period of life.

In the operable group, Meland believes surgery offers the greatest hope of recovery, especially now that the surgical procedures have been standardized. He expects improved radiation results, since radiation treatment is also becoming more standardized. Many cases require both x-radiation and radium.

In a majority of cases, radiation will stop bleeding and pain, and, occasionally, an inoperable case will apparently become operable. This last statement is open to question.

In general, the results of any method of treatment are not satisfactory, as a very high percentage of cases have metastasized before diagnosis has been made. Therefore, any type of treatment is more or less palliative.

JAMES J. CLARK, M.D.

CHOLECYSTOGRAPHY

Some Practical Considerations of Cholecystography from the Roentgenologic Aspect. Eugene P. Pendergrass. Pennsylvania Med. Jour., 42, 787–790, April, 1939.

The author gives the technic of the double-dose administration of dye and also his roentgen technic which is commonly used by most radiologists.

A suggested nomenclature of the findings in cholecystography is given in detail.

A few of the more common points of differential diagnosis are given, and a number of sources of error are mentioned.

JOSEPH T. DANZER, M.D.

THE COLON

Diffuse Polyposis of the Large Bowel, with a Case Report. A. Y. Mason. South African Med. Jour., 13, 237–239, April 8, 1939.

The author describes a case of diffuse colonic polyposis in a 33-year-old Greek electrician who complained of colicky abdominal pains, distention, bloody stools, and diarrhea. A conclusive investigation of the family history of the patient was impossible, but there was suspicion that the patient's father suffered and died of the same disease, which is assumed to be of familial character. Mason points out that, in view of the almost inevitable malignant degeneration of the polypi, treatment is a most urgent problem, be it x-ray deep therapy, colectomy, or surgery combined with fulguration. Although theoretically consenting to treatment, Mason's patient had to date put off the suggested operation. Eight months after diagnosis, he was apparently in good general health, and had succeeded in keeping his symptoms in abeyance by diet and regular daily doses of liquid paraffin.

ERNST A. SCHMIDT, M.D.

Roentgen Diagnosis of *Polyposis coli*. A. Knetsch. Röntgenpraxis, 11, 349–353, June, 1939.

There are two types of polyposis of the colon: one on the basis of an ulcerative or dysenteric colitis; the other the genuine polyposis. In the latter, the inflammatory changes are denied by most authors. It is considered a true neoplastic disease, often hereditary and familial in occurrence. Carcinomatous degeneration is said to take place in from 50 to 60 per cent of these cases (according to Thorbecke and Doering).

A case of extensive polyposis of the colon is described. Two polyps in the rectum had become malignant, as proved by surgery. The combined air-barium enema showed the typical roentgen signs of the disease.

HANS W. HEFKE, M.D.

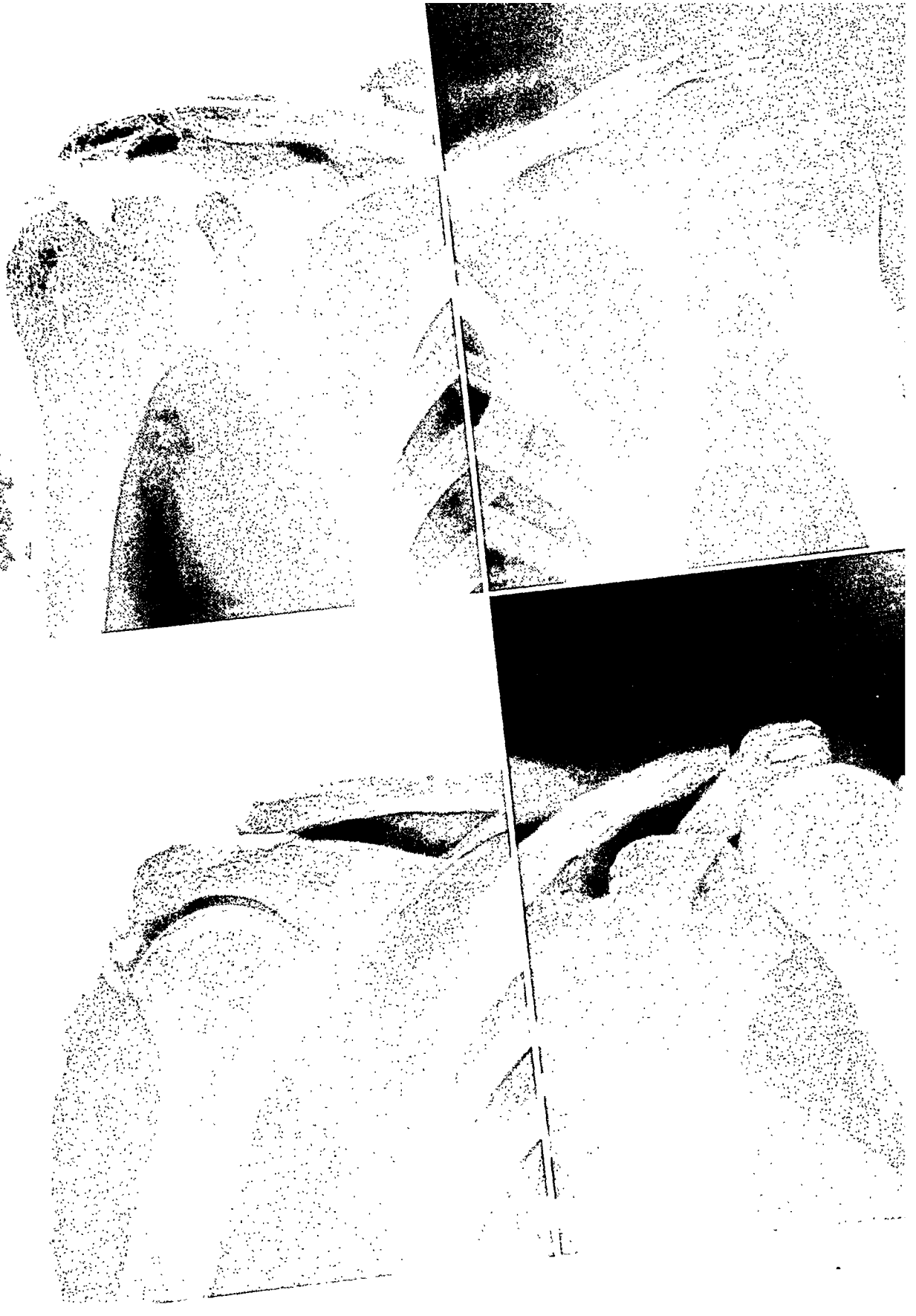
CONTRAST MEDIA

Should the Use of Thorium Dioxide in Hepatosplenography be Abandoned? Enrico Benassi. Radiol. Med., 26, 81–98, February, 1939.

Professor Benassi does not agree with the pessimistic view of some observers who condemn this method of exploring the liver and the spleen because it is too dangerous. He believes that the radio-active substances injected into the organism have not been proven by clinical observations to have a deleterious action on the healthy or sick organism, nor on the evolution of disease, and he affirms that the only justifiable objection that exists to-day against the method is possible delayed damage to the tissues brought about by the feeble radio-activity of the thorium remaining for a long time within the body. This is hypothetical, however.

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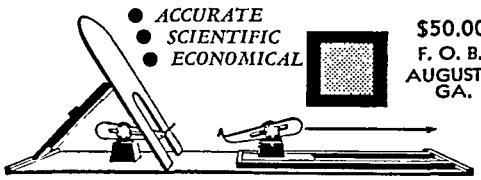
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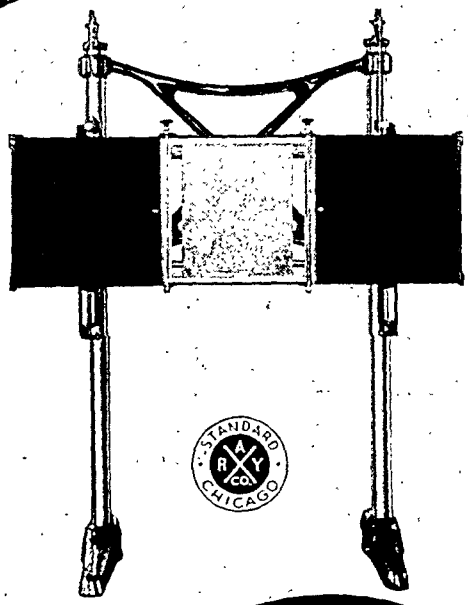
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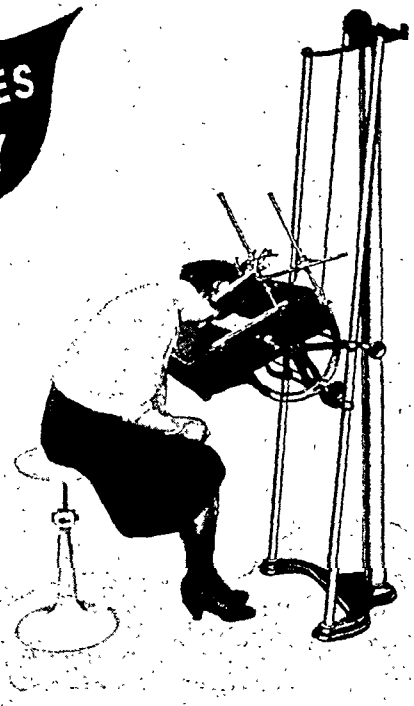
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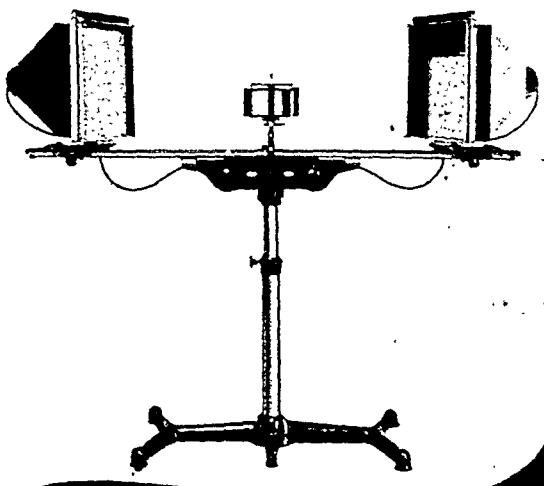
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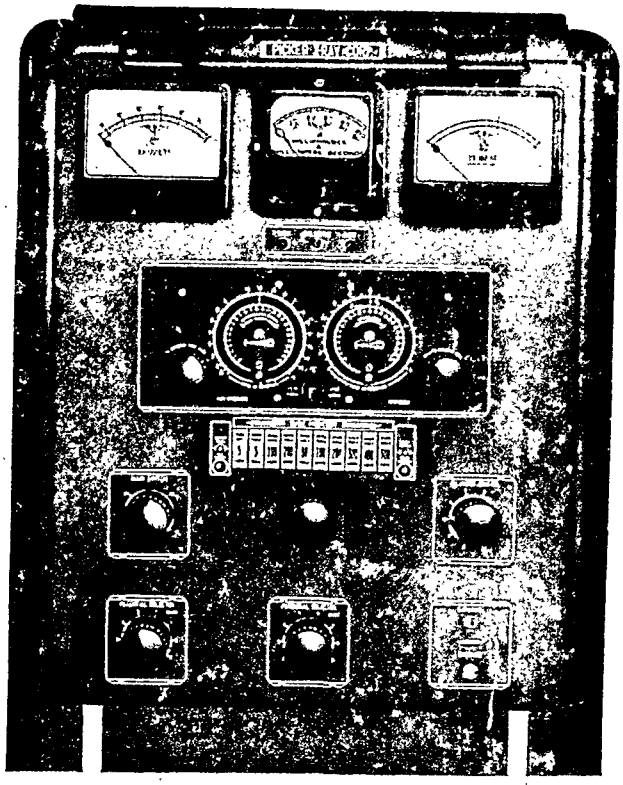
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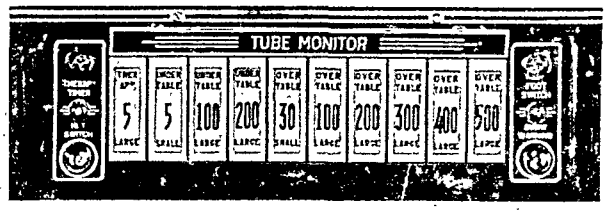
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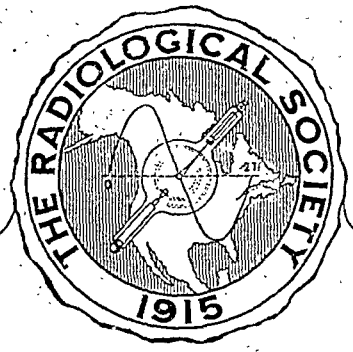
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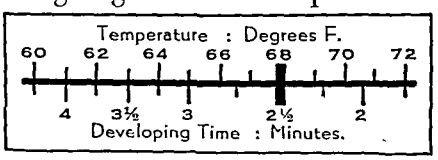


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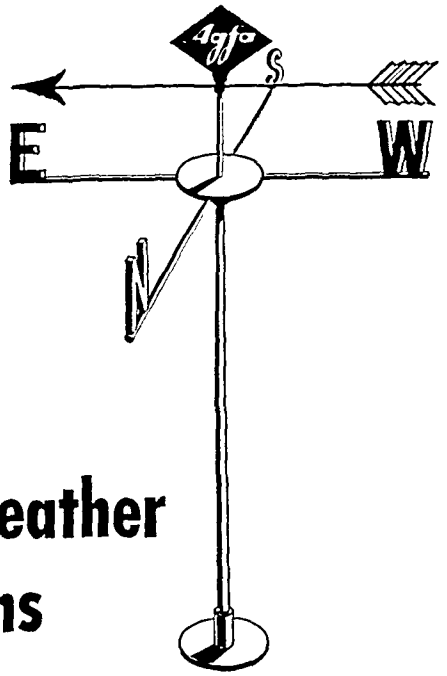
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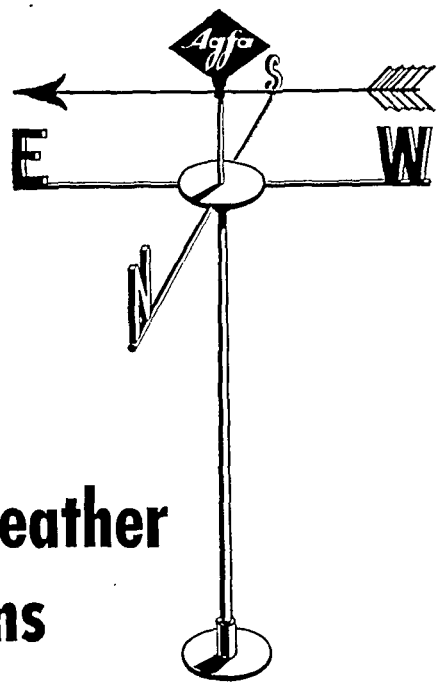
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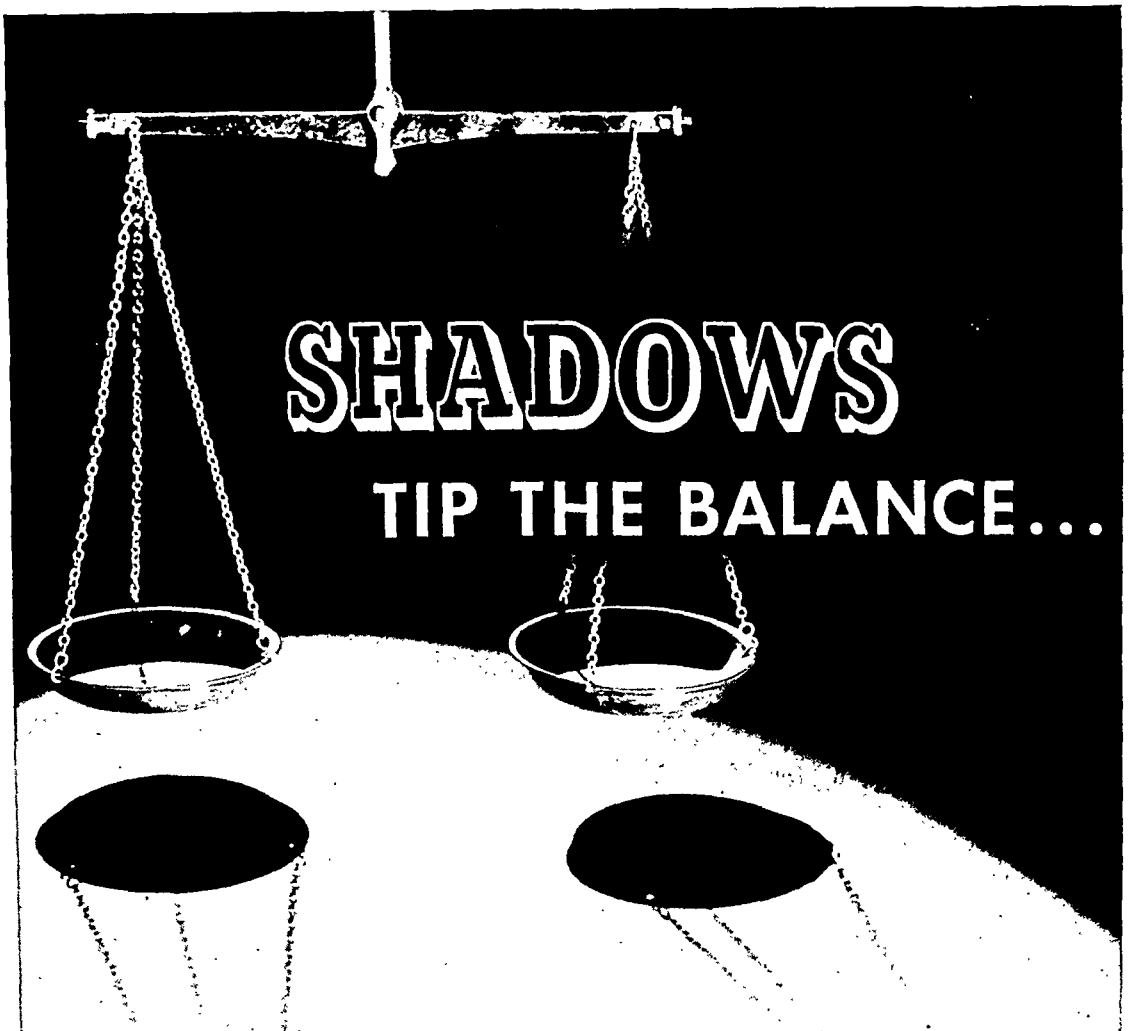
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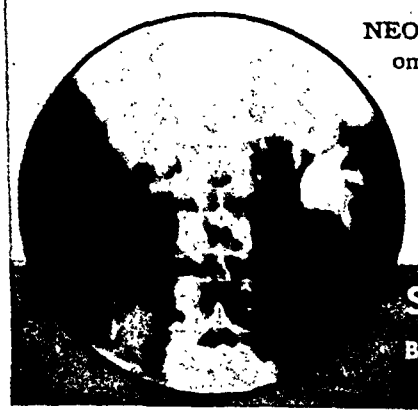
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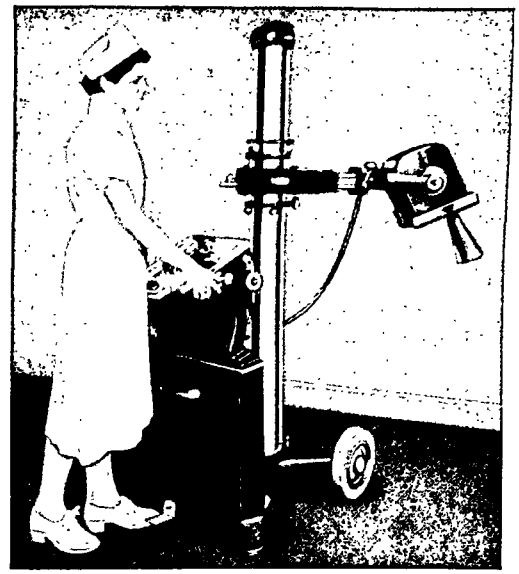
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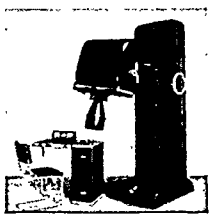
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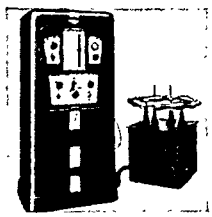
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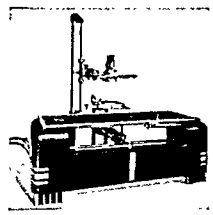
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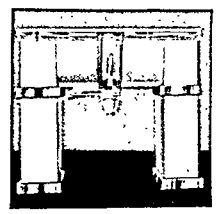
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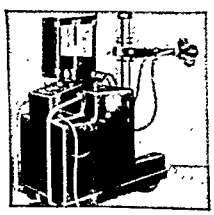
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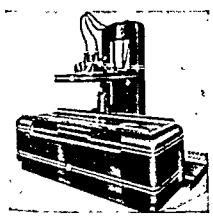
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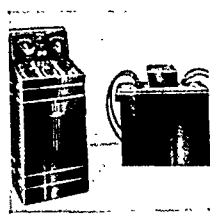
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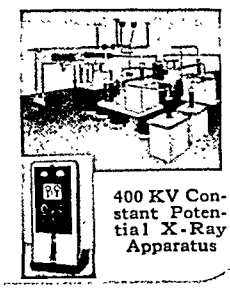
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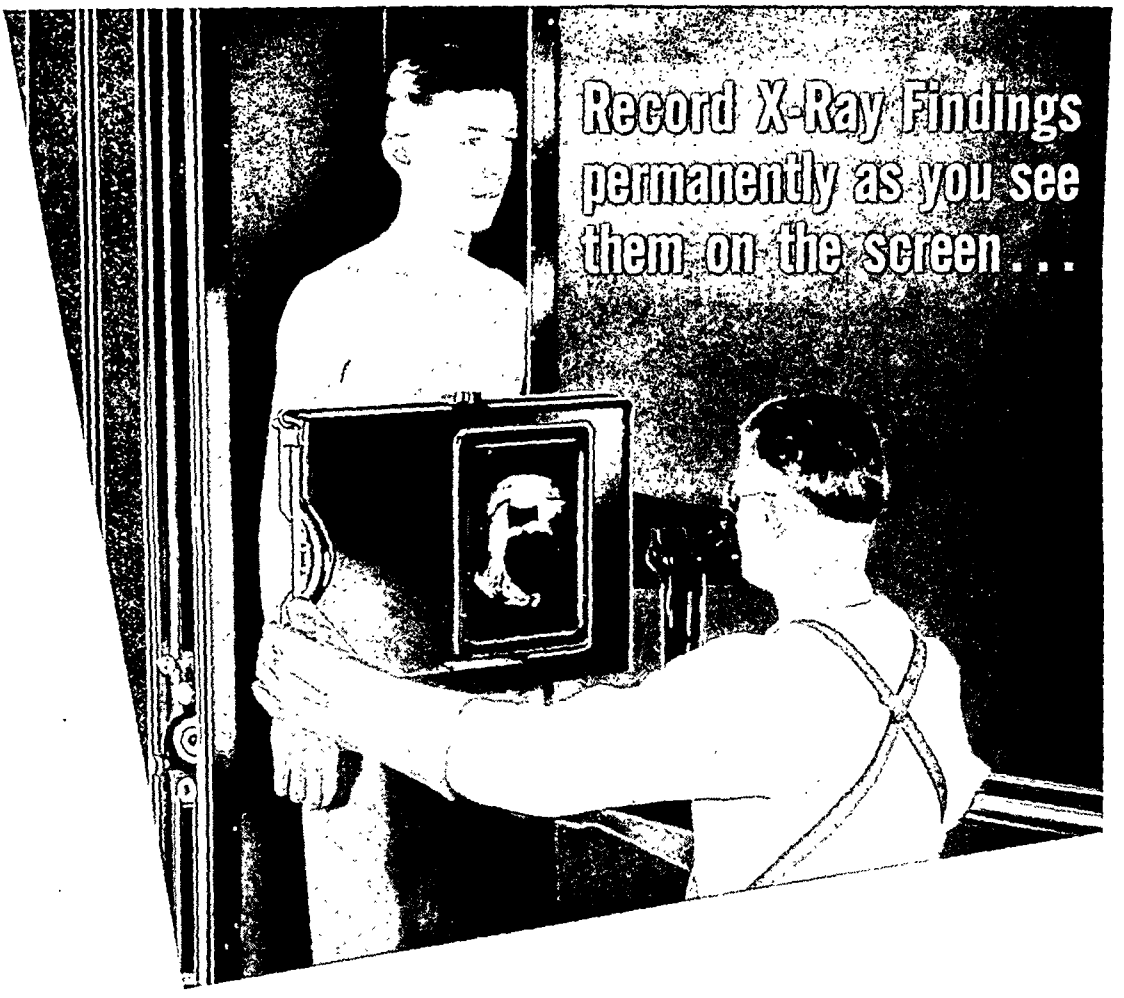
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THE ROENTGENOLOGIC DIFFERENTIAL DIAGNOSIS BETWEEN CANCER AND DIVERTICULITIS OF THE COLON[1]

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From the Roentgenologic Department of the Massachusetts General Hospital

THE difficulty in differentiating diverticulitis of the colon, particularly of the sigmoid colon, from a carcinoma of the bowel is as old as the knowledge of diverticulitis as a disease. Though diverticula of the colon were known to anatomists and surgeons as accidental and rare findings, their clinical importance was not recognized before Graser's demonstration in 1898. He showed the comparative frequency of diverticula, the possibility of inflammatory processes being caused by them, and the difficulty of differentiating the inflammatory mass from a malignant growth of the colon.

In the following decades, a number of excellent clinical (Beer, Moynihan, W. J. Mayo, Telling and Gruner, Judd and Pollock, Spriggs and Marxer, Rankin and Brown, and others) and roentgenologic papers (Abbe, Case, Carman, George and Leonard, Spriggs and Marxer, Berg, Stewart and Illick, Golden, Singleton and Hall, and others) have decreased the differential diagnostic difficulties. Occasionally, however, there are still cases in which diverticulitis leads to such deformities as to make its differentiation from neoplastic disease extremely hard. In such a case one often asks oneself whether it is necessary to enter into an extensive differential diagnostic discussion or whether it would not be much better to transfer the responsibility to the



Fig. 1. Long area of narrowing in the sigmoid due to diverticulitis in a 62-year-old woman who had had attacks of low abdominal pain since the age of 17. Present attacks started six weeks previously with fever and pain. X-ray examination showed multiple diverticula proximal to the narrowing (only one diverticulum visible in this view). The narrowing had not changed essentially three weeks later. At some time during the x-ray examination diverticula were demonstrable within the narrowed area. One year later a perforated diverticulum was drained.

[1] Read before the Twenty-fourth Annual Meeting of the Radiological Society of North America, at Pittsburgh, Nov. 28–Dec. 2, 1938.

surgeon and have him solve the problem by exploratory laparotomy. Unfortunately, the diagnosis is by no means easy or even possible at operation in a fair percentage of cases.



Fig. 2-*A*. Small localized area of constant narrowing due to diverticulitis (*see arrow*). Shelf-like beginning and ending of this area. Preserved mucosal folds. Changing spastic areas proximal to the narrowing. For clinical data, see text.

Moynihan, in 1907, while discussing the mimicry of malignant disease in the large intestine, reported the story of a man with a long history of stomach trouble who had had, on two recent occasions, signs of intestinal obstruction which included fecal vomiting. At operation, a hard inflammatory mass was felt in the duodenum with evident duodenal obstruction. In the ileopelvic colon another hard mass was found which was thought to be a cancer. Since this was deemed to be the more important condition, the duodenal ulcer was left untreated, and the tumor was excised. The opened specimen showed no evidence of cancerous growth, but diverticulitis, the wall of the gut being in some parts one and one-half inches thick.

Mayo, Wilson, and Griffin, in the same year, reported five similar cases and stated: "It is impossible to tell, even by the gross appearance of such a tumor, whether it is inflammatory or malignant."

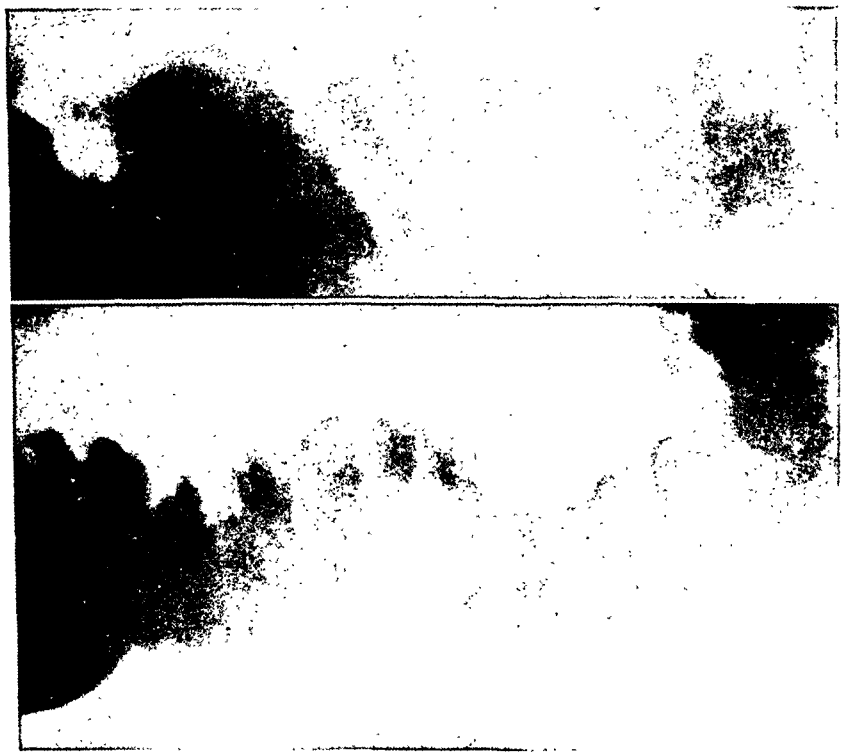
Florian described a patient having intermittent colic, attacks of fever, and intestinal hemorrhages, who had a defect of the sigmoid, shown by x-ray examination, which was called carcinoma. The surgeon had the impression that he had resected a carcinoma, even after he had the specimen in his hand. Microscopic examination alone showed that an area of chronic diverticulitis and not a cancer had been removed.



B *C*

Figs. 2-*B* and 2-*C*. Spot films of the narrowed areas shown in Figure 2-*A*.

Golden reported the history of a patient in whom the family physician had made the diagnosis of cancer of the descending colon based on the findings during a barium enema examination. A hard, non-tender mass was felt in the left lower quadrant. The patient was operated on immediately after his admission to the hospital without any further x-ray studies. At operation the mass was thought to be carcinoma, even though numerous diverticula of the descending colon and sigmoid were noted. The dissection of the specimen proved the mass to be due to diverticulitis and not to cancer.



Figs. 3-*A* and 3-*B*. *A*, Small fibrostenotic area in the sigmoid due to diverticulitis. Note preserved mucosal folds in the area of narrowing. *B*, Three months later the spasticity around the narrowing has improved; the narrowing itself is still present. Clinical data: 13 days previous to the first examination patient was operated on under the diagnosis of acute appendicitis. The operation showed diverticulitis of the sigmoid with perforation.

Among 18 cases of diverticulitis recently reported by Stetton and Abeloff, three had been called cancer at the time of operation. Two of these patients died following the operation. Similar cases have been reported by Telling, Judd, Coombs, and others. In fact, almost any abdominal surgeon can recall cases from his own experience.

A few cases taken from the records of our own hospital may further illustrate this problem.

A man, 54 years old, had been examined outside the hospital and the clinical diagnosis of cancer had been made. The x-ray examination, done on the outside, showed diverticula. At operation a mass was found in the region of the sigmoid. The surgeon was absolutely positive that it was carcinoma and thought it to be inoperable. No x-ray examination was done in our hospital when the patient entered it for a second operation. The mass in the sigmoid was confirmed and, at the time of the operation the differential diagnosis between cancer and diverticulitis was considered. The sigmoid was resected, the

specimen showed diverticulitis, with no evidence of cancer. The patient died post-operatively.

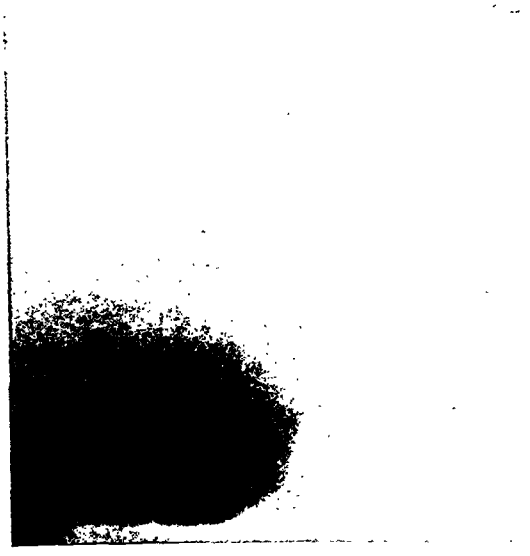


Fig. 4-A. Obstruction due to diverticulitis of the sigmoid. The film shows the tip of the barium enema where there is no tumor visible.

Examination of a woman, 36 years of age, with a nine-day history of a "bloated feeling" in the abdomen showed a large, slightly tender mass arising from the pelvis. At laparotomy a large fibroid of the uterus was removed. As the operation was being completed, a hard nodule was felt in the sigmoid. This seemed to be producing some constriction, and, as there was an implant on the peritoneal surface which looked malignant, a Mikulicz resection of that loop was done. The resected specimen showed a single diverticulum extending into the mesenteric attachment, surrounded by a fibrous mass, 3 cm. in diameter, and two firm glands close to the peritoneal surface of the mesentery. Microscopic examination proved the presence of chronic diverticulitis. There was no evidence of malignancy.

All these reports demonstrate dramatically how difficult—or even impossible—it may be to differentiate between diverticulitis and cancer during the operation.

Inasmuch as resection of diverticulitic masses is connected with a high operative mortality and is indicated in comparatively rare instances, the necessity of making the correct diagnosis before opening the abdomen is obvious. Clinical data in regard to age, weight loss, pain, stool habits, temperature, and blood picture are of some, though usually not decisive, importance. They will not be discussed in this paper (see Telling and Gruner, Judd and Pollock, Spriggs and Marxer, Case, Rankin and Brown). The presence of blood in the stools is strongly in favor of cancer in a questionable case, and I agree with those authors who advise looking for an additional source of hemorrhage if, in a case of diverticulitis, blood is found.

Unless proctoscopy demonstrates a cancer the responsibility of differentiating new-growth from diverticulitis rests heavily and almost exclusively on the roentgenologist. It is the main purpose of the present paper to stress this sometimes uncomfortable but unescapable responsibility which is recognized by competent clinicians, though the necessary consequences inherent to such a responsibility are not always followed by either roentgenologist or clinician in the handling of such a case.

The pathology of diverticulitis has been described in excellent reports (see Telling and Gruner, Spriggs and Marxer, Case). It will be mentioned here only inasmuch as it seems necessary for the differential diagnosis.

The inflammation within and around diverticula, probably due to stagnation of fecal material within them, leads to swelling of the mucosa. It is possible that some of the swelling is due to lymphostasis produced by sclerosing inflammatory processes of the mesenteric glands, as suggested by Berg, Pohl, and Fleischner. The course of the disease may be acute, like appendicitis, and may lead to perforation of a thin-walled diverticulum, producing either diffuse—or, more frequently—localized peritonitis with formation of an abscess. The anatomic changes produced by the more or less chronic form of the disease, in which infiltration of the wall of the intestine around the diverticulum is the most striking

feature, are of greater differential diagnostic interest. This inflammatory infiltration—which may involve all the layers of the wall below the mucosa—may recede by resorption of the inflammatory elements, or it may result in secondary fibrosis leading to the production of a firm mass within the wall of the intestine beneath the mucosa. Such thickening of the wall up to two inches has been described. The adjacent mesentery may be involved in the inflammatory and fibrotic process, thereby adding to the size of the mass.



Fig. 4-*B*. Examination eight days later shows the obstruction to be incomplete. Multiple diverticula are demonstrable. There is swelling of the mucosa of the sigmoid with localized narrowing. Clinical data: 45-year-old woman; history of crampy pain and diarrhea without blood for the last two weeks. Tender mass in the left lower abdomen, which had decreased in size at the time of the second examination.

The early inflammatory changes of diverticulitis show a more or less spastic portion of the intestine with wide transversely arranged folds producing a sawtooth or an accordion-like appearance, as first described by George and Leonard. This picture may simulate local inflammatory processes other than diverticulitis and can be produced also probably by destruction of the draining mesenteric lymphatics (Berg, Fleischner), but it cannot be confused with intrinsic cancer.

Abscesses produced by perforation into a walled-off area may be seen outside the bowel, associated usually with spasticity and other signs of diverticulitis. It is, however, important to state that the irritation may completely disappear, although a large inflammatory mass, with abscess formation, may still be present. If in such a case the barium does not enter the abscess cavity, an almost normal picture of the gut may result.

Increasing inflammatory swelling produces cushion-like projections, separating long-necked diverticula from each other. This picture has been described and illustrated extensively in the literature (Case, Spriggs and Marxer, and others). The process results in a bizarre, fringed contour, which, in spite of its marked degree, gives a deformity so characteristic that it cannot be confused with intrinsic cancer.

Differential diagnostic difficulties arise if inflammatory masses are arranged with fair evenness throughout the wall of the



Fig. 5. Mucosal picture of a small area of cancer at the junction of the descending colon and sigmoid. Complete destruction of the mucosal relief within the area of cancer (*see arrows*). Swelling of the mucosal folds proximal to the cancer.



Fig. 6. Localized narrowing of the sigmoid (*see arrow*) in a patient with diverticulosis of the descending colon. (Cf. Figures 7 and 8.)

intestine beneath the mucosal surface (Fig. 1). The resulting concentric constriction usually has cone-shaped ends and extends in most cases over a fairly large part of the intestine. The wall of the intestine in the region of the cone-shaped beginning and ending of the lesion is usually not absolutely rigid, as can be seen by the changing lumen during any one examination. The constricted area itself may or may not change in size, depending on the degree of rigidity. The mucosal folds are preserved and may or may not be swollen; the size of the lumen in the constricted area itself may vary, from week to week, depending on the amount of cellular and serous, or fibrous elements.

The mimicry of cancer may, however, be still more marked, as the following case shows. A man, 55 years old, entered the hospital complaining of loss of weight and of rumbling gas in the abdomen. A questionable mass was felt in the left lower quadrant. The clinical diagnosis was carcinoma of the sigmoid. The barium enema showed a localized narrowing about two centimeters in length (Fig. 2). The narrowed area was not cone-shaped, but had a shelf-like margin on both ends, an appearance usually considered to be characteristic of the elevated edges of an intrinsic tumor. The area was completely rigid, and had not changed at a second examination done two days later. It did not relax with amyl nitrite. Proximal to this area there were a number of diverticula as well as definite spasticity of the gut. After inhalation of amyl nitrite this spastic area relaxed and the absolutely rigid region distal to it became even more striking. The rugæ in the suspicious area were, however, definitely preserved. In other words, the patient had, in addition to characteristic diverticulitis, a lesion which had all the signs of an intrinsic mass, such as absolute persistency in size and appearance. It had sharply defined edges and it was small in size, but the mucosa was not destroyed. The lesion, therefore, had to be localized within the wall of the bowel, but beneath the mucosa, and the diagnosis of diverticulitis without cancer was made. The resected specimen showed the narrowed area to be produced by fibrous masses within the wall of the intestine covered by normal mucosa. The roentgen-ray appearance of this lesion differed from that of a small cancer only by the appearance of the mucosa.

Another similar case in which the edge was not quite as sharp as in the preceding

case illustrates the same problem (Fig. 3-*A*). The lesion involved predominantly one side of the sigmoid. Three months later, the characteristic diverticulitis proximal and distal to the lesion had lost its inflammatory character—draining of a peridiverticular abscess had preceded the first examination by thirteen days—while the small fibrostenotic area was essentially unchanged (Fig. 3-*B*). It is characteristic of this type of lesion that, in the presence of localized rigidity and of all the evidence of a lesion within the wall of the bowel, the mucosal folds are preserved, although these folds may have a rather fixed arrangement such as is unusual for the constantly changing pattern of the normal colon.

The most difficult problem in differential diagnosis arises in cases with complete obstruction. It is obvious that it is difficult to diagnose a lesion when it is not outlined by barium. Case described, many years ago, the demonstration of the inflammatory mass in diverticulitis by the presence of an unusually long distance between the small intestinal loops and the brim of the pelvis on the left side. In cases of obstruction this mass may be clearly outlined by the air-filled small intestinal loops. It does not, of course, enable one to make a definite differentiation between inflammatory and cancerous mass. Usually all our efforts have to be concentrated on an attempt to bring at least traces of barium beyond the obstruction. The inhalation of amyl nitrite may occasionally be helpful. It is of greater importance to wait patiently for a few drops to pass through the stenosed area without over-distending the rectum, and to replace constantly any barium which may be emptied through the relaxed sphincter.

Occasionally films made after evacuation show barium beyond the point of obstruction, which was apparently transported upward during defecation. Sometimes *careful* inflation of the colon with small amounts of air (Fischer, Weber) may outline the stenosed area. If all these efforts are in vain, the appearance of the colon at the point of obstruction is often helpful. If a cancer is the cause of the obstruction, one will usually be able to see the surf-



Fig. 7. The same patient as shown in Figures 6 and 8, showing the extensive diverticulosis of the descending colon. The area of narrowing is less distinctly visible in this projection (*see arrows*). A loop of small intestine is partially superimposed over the sigmoid.



Fig. 8. The same patient as shown in Figures 6 and 7. Post-evacuation film shows definite destruction and ulceration in the area of the narrowing (*see arrows*). This patient had diverticulosis of the descending colon and cancer of the sigmoid Operation.

of the obstructing tumor, particularly during fluoroscopy or on films made after evacuation. In diverticulitis no definite tumor surface is visible at the point of obstruction; diverticula may or may not be demonstrated (Fig. 4). The diagnosis in such a case will usually be obstruction without demonstrable tumor mass. This is a diagnosis which is by no means pathognomonic of diverticulitis but which is very much against the presence of an intrinsic tumor. If a positive diagnosis is absolutely impossible, re-examination after one week of bed rest and bland diet may be helpful. If such a delay is not justifiable from the clinical picture or if the second examination again shows complete obstruction, a simple cecostomy without any attempt to explore the sigmoid may be the treatment of choice. It will relieve the obstruction. Another barium enema, one or two weeks later, very frequently will show the obstruction to be incomplete and will then enable one to recognize the pathologic process. This procedure will cause no loss of time if the lesion should turn out to be cancer, inasmuch as colostomy usually represents the first part of a two-stage resection of a sigmoid cancer, and it is, at times, good treatment for a stenosing diverticulitis.



Figs. 9-*A* and 9-*B*. *A*, Localized narrowing of the sigmoid (*see arrows*) in a patient with diverticulitis. *B*, Spot film shows destruction of the mucosa with ulceration in the area of the narrowing (*see arrows*) as evidence of cancer. Definite diverticulitis proximal and distal to the small cancer. Operation.

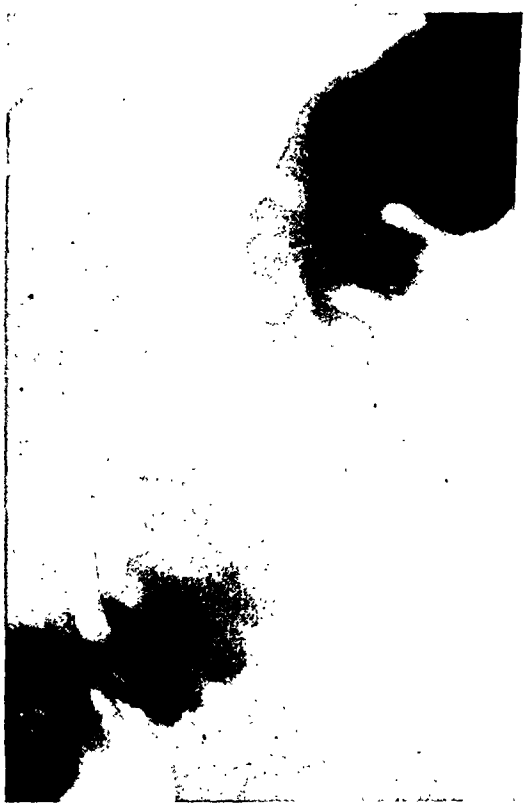


Fig. 10. Intramural extramucosal recurrence of cancer in the area of resection of a cancer of the lower descending colon. At operation a mass was found involving the wall of the bowel. The mucosa showed slight wrinkling, but it was not involved. Note the characteristic appearance of an intrinsic lesion with preserved mucosal folds.

In cancer of the colon the defect produced by the tumor shows sharply defined margins. Instead of the normal mucosal folds one sees the irregular tumor surface, usually with ulceration (Fig. 5). Very commonly the colon proximal and distal to the cancer appears normal. Spasticity and swelling of the mucosal folds may, however, be present, particularly proximal to the lesion as in diverticulitis. Diverticula may be present proximal and distal to the tumor and may complicate the diagnosis, particularly in small lesions. Figures 6 to 8 show the colon of a patient who had been treated for years for constipation. The examination showed multiple diverticula in the colon, with localized narrowing in the sigmoid. There was definite loss of the mucosal pattern with evidence of ulceration in this region, and the diagnosis of cancer with diverticulitis was, therefore, made. At operation the tumor was found to be inoperable due to liver metastases.

Not only simple diverticulosis but also definite diverticulitis may be present, together with a small cancer (Fig. 9). In other words, the presence of diverticula or diverticulitis does not help in the differential diagnosis between cancer and diverticulitis in a questionable, localized, narrowed area. One should hesitate to diagnose diverticulitis in the absence of demonstrable diverticula, but their presence does not help one way or the other.

In the description of the roentgenologic appearance of diverticulitis, I have stressed the importance of the recognition of localized fibrotic masses in the wall of the intestine. It is obvious that any intramural extramucosal tumor will produce a similar picture. A large variety of tumors may metastasize around the sigmoid and into its wall. Ovarian carcinoma and endometriosis do so most commonly. The differentiation of these from diverticulitis and primary carcinoma of the sigmoid goes beyond the scope of this paper and shall, therefore, be discussed only briefly. If such a tumor has destroyed the mucosa, a differentiation from primary carcinoma of the sigmoid is usually impossible. If it surrounds the intestine without involving its wall, it produces a pressure defect in an otherwise normal colon, thereby differentiating it from intrinsic carcinoma and from the majority of cases of diverticulitis. The localized intramural extramucosal tumor nodule may be differentiated from a similar diverticulitic lesion by its shape and by the absence of diverticula.

A roentgenologic differential diagnosis between extrinsic tumor masses and diverticulitis may become impossible if the masses have produced obstruction and incidental diverticula are present. Fortunately enough, however, it is usually much easier for the clinician to differentiate these lesions from diverticulitis, judging by his clinical examination and by the findings at operation. A pre-operative roentgenologic diagnosis is, therefore, not so badly needed as in the differential diagnosis between diverticulitis and intrinsic carcinoma of the colon.

If a resected cancer of the colon recurs locally in the wall of the intestine beneath the mucosal surface, it may also produce a picture of an intramural extramucosal lesion (Fig. 10). The history of such a case readily differentiates it from localized intramural inflammatory fibrosis.

SUMMARY

A differential diagnosis between cancer and diverticulitis of the colon is, in a fair percentage of cases, difficult or impossible during operation. The treatment of choice is different in most cases and a pre-operative diagnosis is, therefore, necessary. The roentgenologic differential diagnosis between the two diseases is easy in most cases, difficult in some, and impossible in a few.

Particular stress is laid on the differential diagnosis of localized fibrostenotic diverticulitis from cancer. In some of these cases, the characteristic picture of an intramural extramucosal lesion results. In such cases, the preservation of the mucosal folds is the only finding which differs from the appearance seen in a carcino-

matous lesion. It is possible that cancer produces occasionally a slightly fold-like picture. Usually, a differentiation of such an area from normal rugæ is not difficult. A warning against the indiscriminate interpretation of the rugal pattern seems necessary, however, particularly in cases in which the diagnosis rests exclusively on such interpretation.

The presence of diverticulosis or diverticulitis proximal or distal to a questionable narrowed area of the colon is of little or no value in differentiating cancer from diverticulitis.

Diverticulitis without demonstrable diverticula is rare.

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DISCUSSION

James T. Case, M.D. (Chicago): We have heard and seen a detailed presentation which seems to be almost perfectly complete, for which the author deserves commendation. The problem is a very real one and, even though he has shown a collection of instructive cases, I still feel that there will be many cases in which the diagnosis will be up in the air, after trying all of these devices of examination, and time alone will prove the lesion not cancerous.

The mucosal demonstration is a valuable means of differentiation and, as Dr. Schatzki has emphasized, if one decides upon the introduction of air to aid that purpose, it had best be done with extreme care. I notice that in one of his cases the colon was apparently over-distended with air. I would warn against using over-distention of the colon in doing air-enema combined studies. I have seen two cases of rupture of the sigmoid in the presence of diverticulitis, the rupture resulting from manipulation of the sigmoid and distention with a relatively small amount of air.

The speaker's recital of a case of a single colonic diverticulum in a patient who had had a pelvic operation, the tumor being found at the time of the operation, parallels a case of my own in which removal of a pelvic tumor was undertaken without preliminary x-ray study. At operation there was found also a tumor of the sigmoid, which was removed because it looked so surely cancerous; afterward it was found to be a diverticulitis with a single diverticulum.

I would like to report [the speaker showed slides] several cases to illustrate my remarks; not to show any difference of opinion with Dr. Schatzki but perhaps to emphasize what he has said.

My first is a typical case of carcinoma of the sigmoid in the presence of diverticula. This shows definitely the clean-cut, shelf-like defect of which Dr. Schatzki spoke—a clean-cut shelf at the distal end of the tumor, as shown by the barium enema, a finding always suggestive of cancer.

The next patient had a tumor of the sigmoid involving the bladder, which necessitated a resection of the fundus of the bladder along with the tumor. It was definitely carcinoma but in these films we see a diverticulosis of the sigmoid rather than a diverticulitis—perhaps the differentiation is more definite than in the presence of diverticulitis.

Next is the case of a dentist whose barium enema gave the findings of an irregular filling defect in the lower sigmoid, the differential diagnosis between carcinoma and diverticulitis not being easy. In the immediate films the appearance was such as to indicate carcinoma but in films made a few hours later, several small dots of barium developed near the lesion and gave the clue that we dealt with diverticula. Even at operation the surgeon could not decide, and he interrupted his surgery long enough to call me up for my final opinion as to cancer or diverticulitis. I voted for the latter and only a colostomy was done. Now, seven years later, the patient continues his dental work, but he has developed an ileocolic fistula, short-circuiting his inguinal colostomy and the stricture.

Ever since our earliest work in the colon we have emphasized the importance of making films of the sigmoid in the oblique projection, and, in cases of suspected diverticula, of making delayed films to allow time for the opaque material to work into the diverticula.

This case is a little more difficult—a man of 65 with a palpable mass in the left side of the abdomen and a very high grade of obstruction. I show you two sets of films—one without air injection, others with a little air carefully given. Because the mucosal folds were preserved throughout the lesion and because the delayed films showed penetration of barium into the diverticula, we were able to recognize the diverticulous nature of the lesion. Three years have passed under non-operative management and, although a certain grade of obstruction persists, by dietary and other means he manages to get along.

Whenever there is a question of diverticula, I always feel it is necessary to follow up the barium enema study by a study a few hours later or the next day to make sure we are not overlooking the diverticula.

MAURICE FELDMAN, M.D. (Baltimore, Md.): The diagnosis of a double lesion is always a perplexing one when it comes to pathology in the colon. In a differential diagnosis between carcinoma and diverticulitis, there is considerable difficulty and in some cases, of course, as Dr. Schatzki has pointed out, the diagnosis cannot be made with certainty.

In all these cases it must be borne in mind that the two conditions may coexist. The coexistence of carcinoma and diverticulosis occurs in a small percentage of cases. It seems to me that the mucosal relief film is the most important method of eliminating the two conditions. Even here one may find great difficulty. It is well known that in diverticulitis the size of the tumor has a tendency to change,

and this changing of the size of the tumor is a very important clinical fact.

Another important fact is that in diverticulitis the obstruction is rarely complete. It has been my experience that the law of averages is against the double lesion in those cases which show a diverticulosis, and in cases in which we find a localized lesion in the sigmoid, the law of averages would indicate the possibility of a diverticulitis although in a small percentage of cases this law is at variance.

Clinically, the picture is somewhat different, too. The mass is sausage-shaped and sometimes one may feel the mass while at other times it may not be felt at all.

Blood in the stools is ordinarily not seen in diverticulitis unless there is some complication such as ulcer or polypi. The presence of blood is variously reported as between 3 and 25 per cent of cases. In our experience we have seen the presence of blood in only a very small per cent.

DR. SCHATZKI (*closing*): I want to thank Dr. Case and Dr. Feldman for their discussions. It was a great honor for me to find that a man with the experience of Dr. Case did not disagree too much with me.

There are two points which I thought I had to discuss again when I saw Dr. Case's slides. One, in regard to the shelf. I do not wish to be misunderstood. The shelf is by all means much more common in carcinoma than it is in diverticulitis, and if I should see a shelf, particularly on both sides of the lesion, I would hesitate very long before all the other factors which, in the examination of a given case, are against cancer would make me exclude cancer.

Another thing about which I do not want to produce a misunderstanding is the importance of the demonstration of diverticula. The presence of diverticula does not help very much in the differential diagnosis between the two diseases but the absence does, and, therefore, it is so extremely important to prove the absence. That is often difficult. Therefore, we have to be careful before we make the statement that diverticula are not present.

The presence of diverticula may become important if they are present within the narrowed area because I think it is rare—I do not know if it occurs at all—that we find them right within a narrowed cancerous area.

THE HEMATOLOGIC DIAGNOSIS AND ROENTGENOLOGIC TREATMENT OF MYELOGENOUS LEUKEMIA[1]

By WALTER C. POPP, M.D., Section on Therapeutic Radiology, *and* CHARLES H WATKINS, M.D., Division of Medicine, The Mayo Clinic, *Rochester, Minnesota*

THE importance of an accurate hematologic diagnosis in myelogenous leukemia, particularly in relationship to the phase of the leukemia, cannot be overemphasized. The relative activity of any variety of leukemia is and should be of vital concern to the roentgenologist. For that reason we thought it worth while to present this subject from the viewpoint of the hematologist as well as that of the roentgenologist. We will present the hematologic pictures of four different phases of myelogenous leukemia and make suggestions as to both roentgenologic treatment and a method of follow-up examination which we think permits better control of a patient who has leukemia than is offered by sporadic treatment.

Myelogenous leukemia may be classified into four general types based on clinical and hemacytologic criteria. Diagnosis of acute leukemia may be made without difficulty. The disease usually has a rapid onset associated with fever, anemia, usually hemorrhage from the mucous membranes, petechiæ and purpuric manifestations in the skin, and, usually, a progressive enlargement of the spleen. The hemorrhagic phenomena may be so outstanding as to suggest a primary hemorrhagic disease, particularly purpura hemorrhagica. If the type of leukemia is aleukemic or leukopenic, it may be confused with acute aplastic anemia. Purpura hemorrhagica is usually readily distinguishable by the well known tendency of this disease to occur in remissions and exacerbations, in a relatively mild degree, and frequently over a relatively long period before an acute exacerbation occurs. Regeneration of erythrocytes is usually good and very little, if any, immaturity of the myeloid leukocytes will be found in the stained blood smear. Aplastic anemia frequently may be distinguished from acute leukemia by the absence of a palpable spleen. Examination of the stained smear reveals decreased regenerative activity of the myeloid leukocytes, rarely ever any slightly immature forms, relative lymphocytosis, thrombocytopenia, and a decreased number of reticulated erythrocytes. Acute myelogenous leukemia usually presents an elevated leukocyte count, but even if the leukocyte count is normal or if a leukopenia is present, examination of a stained blood film will establish the diagnosis. In acute myelogenous leukemia the predominating leukocyte is the myeloblast or stem cell, together with slightly more mature stages in the development of the myeloid leukocytes, leukoblasts, and promyelocytes. In any case in which this marked degree of immaturity associated with the clinical features previously listed is presented, the diagnosis of acute leukemia is warranted. In our experience treatment of this condition has been of no avail.

Subacute myelogenous leukemia usually is less dramatic in onset than acute leukemia, although, as the disease progresses, hemorrhage may occur as the result of reduction in the number of blood platelets in the peripheral circulation. Fever and anemia are usually associated with hemorrhage. The spleen in most cases is palpable, and examination of the stained blood film shows the predominant cell to be the leukoblast, with relatively few myeloblasts or stem cells, numerous promyelocytes and occasional myelocytes in more mature forms. It is most important to establish the diagnosis of subacute myelogenous leukemia for, in most instances, we feel that treatment is contra-indicated and it

[1] Read before the Twenty-fifth Annual Meeting of the Radiological Society of North America, at Atlanta, Dec. 11–15, 1939.

is rather probable that most instances of subacute leukemia are really mild forms of the acute process, which means that roentgen therapy should be avoided. On the other hand, an occasional instance of the condition may present a rather high proportion of immature cells, associated with an almost equal number of relatively mature leukocytes, and a moderate reduction in blood platelets. In spite of the rather rapid onset of the disease, it will respond rather well to small doses of roentgen rays.

Chronic myelogenous leukemia usually is characterized by a gradual onset of anemia, a slowly progressive increase in myeloid leukocytes, and an insidious enlargement of the spleen. Many times the initial complaint of the patient is that of a tumor in the left side of the upper portion of the abdomen. For this reason it is often difficult to determine how long the disease has been present in a given instance. Examination of the stained smear discloses a marked increase in the number of myeloid leukocytes, the predominant cell being the mature neutrophil; metamyelocytes and myelocytes will be rather numerous. Cells that are more immature will be present but, when they are compared with the late phases in the development of myeloid leukocytes, myeloblasts, leukoblasts, and promyelocytes they will not be numerous.

The leukopenic or aleukemic phase of myelogenous leukemia may occur in any of the types of leukemia previously described, the final diagnosis resting upon a detailed study of the stained blood film. Because of the low leukocyte count, roentgen therapy must be administered with great caution and under no circumstances should patients who have the acute or subacute types of the disease receive roentgen therapy.

A number of methods of treatment for myelogenous leukemia are in use to-day, the more popular being irradiation of the spleen, long bones, mediastinum, and, more recently, the use of small amounts of radiation to the trunk at a relatively long target-skin distance, the so-called x-ray bath. We employ the method of treating the spleen, using a definite number of fields which, as a rule, are relatively small, the dosage being varied according to the phase of myelogenous leukemia under treatment. Our experience, based on about 350 cases, has led us to favor the use of moderate voltage (135 kv.) technics over all others we have tried.

The area of the spleen is divided into nine fields of approximately equal dimensions and of such size as to cover the entire spleen. There are four anterior, one left lateral, and four posterior fields. The four posterior fields correspond with the levels of the four anterior fields. These fields are numbered from one to nine, a procedure which provides nine fields of exposure over the spleen, thus permitting as many as nine treatments should that many be indicated, without repetition of exposure to any portion of the skin in a single course of treatment. Before treatment, a complete hematologic study is made of the erythrocytes as well as the cytologic aspects of the leukocytes. Should severe anemia be present, a transfusion of blood may be given. If only moderate anemia is present, transfusion is not indicated, since the majority of patients who have moderate anemia have a relative increase in erythrocytes as the leukocytes decrease in number. After the phase of myelogenous leukemia has been determined, the course of treatment is begun, and should first be directed to one of the fields of treatment near the lower edge of the spleen. On the following day a leukocyte count is made and treatment is continued, provided there has been no radical decrease in the total count. This method is followed daily until the leukocyte count reaches what seems to the hematologist to be a satisfactory level. This level, of course, varies considerably with the phase of the leukemia under treatment. For example, if the treatment is being carried out for chronic myelogenous leukemia, the number of leukocytes can be reduced more quickly than would be possible if a subacute or

aleukemic leukemia were present. If the count seems to have decreased too radically, treatment should be interrupted for from 24 to 48 hours so that the significance of such a decrease can be evaluated. We believe that a course of treatment should be carried out in a relatively short period of time, so that the patient can be allowed to maintain himself with no treatment for as long as possible; treatment is resumed when the leukocyte count begins to show a definite and persistent increase, particularly in the immature cells.

It is not necessarily true that the higher the count, the more treatment is necessary. It has been our experience that patients having relatively low counts of from 75,000 to 100,000 cells need more treatment to produce the desired effect than do patients who have a count of from 250,000 to 300,000 cells. In treating patients who have myelogenous leukemia, the leukocyte count should not be lowered beyond certain flexible values depending, of course, on the original count. If, for example, the original count in the presence of chronic myelogenous leukemia, before roentgen treatment, is approximately 75,000 cells, the course of treatment should not be carried beyond a point at which the count reaches approximately 25,000. On the other hand, if the original count is approximately 250,000, treatment should not be carried beyond a point at which the count reaches 50,000 to 60,000 cells. The reason for this precaution is that, after the patient has completed a course of treatment, the leukocytes continue to decrease in number for as long as a month or more and, should the treatment be carried too far, a dangerous leukopenia may develop. We have found that leukocyte counts which have been reduced to about 50,000, after a period of three weeks, will usually reach a normal level. We feel that the general condition of patients is better if they have a slight leukocytosis rather than a leukopenia. With a course of treatment being administered in a relatively short period averaging seven to nine days, it is easier to control leukemia and such a course facilitates additional treatment when resumption of such treatment becomes necessary. The use of a large number of small fields, in addition to permitting a fairly large number of treatments to be administered with little or no dermal reaction, causes almost no discomfort to the patient. It is not at all uncommon for patients to receive daily treatments of this type without experiencing any systemic reactions whatever.

In the presence of acute myelogenous leukemia there seems to be little that can be done therapeutically except to give transfusions of blood and provide supportive measures, and our experience has been the same as that of other workers in that roentgen treatment for this condition is contra-indicated except with rare exceptions.

In treating patients who have subacute myelogenous leukemia, the plan of treatment using nine fields is adopted as suggested previously. The dosage per field should, in our experience, not exceed 75 to 80 r measured in air. This is a small amount of radiation, but it has been sufficient gradually to alter the leukocyte count characteristic of this type of myelogenous leukemia. Treatment is administered daily until a satisfactory result is obtained. The treatment should be interrupted when the leukocyte count has reached a level of approximately one-third the original count. For example, if the original count was 150,000 cells, the course of treatment should be interrupted when a value of not less than 50,000 has been reached. Because of the radiosensitivity of the cells present in this type of myelogenous leukemia, there is great risk in using stronger doses because destruction of the leukocytes may get out of control, with serious consequences.

Chronic myelogenous leukemia is probably the least difficult form of leukemia to treat. The general condition of patients who have this type is usually better than that of patients who have the other types of myelogenous leukemia.

Roentgenologic treatment using nine

fields is arranged as previously suggested. The dosage employed is approximately 225 r per field. Daily sessions of treatment are employed so long as the count does not decrease radically. When the number of leukocytes reaches the desired level treatment is interrupted and the patient is allowed to go without treatment until the count shows a definite tendency to increase. The level to which the count should be allowed to fall is variable. The original level of the count is important in determining the level to which it should be reduced. For example, if the original count before treatment was 300,000, treatment should be stopped when it has reached a level of from 50,000 to 75,000. If the original count was 150,000, treatment should be carried on until the count has been reduced to a level of approximately 25,000 to 35,000. If the original count was 75,000, it can safely be reduced to a level of approximately 20,000 to 25,000. In the presence of this type of leukemia the leukocyte count should not be reduced to less than 20,000 unless the original count was not more than 20,000. With an original count of about 20,000, treatment may be administered until a level of about 10,000 is reached.

In the treatment of a patient who has aleukemic myelogenous leukemia a somewhat different problem faces the roentgenologist. Such a patient has a spleen which varies in size from mere palpable enlargement to a size sufficient to fill the left upper quadrant of the abdomen. The leukocyte count has variations from leukopenia of 2,000 cells to leukocytosis of 10,000 cells. In this type of myelogenous leukemia, treatment must be carried on with small doses and small areas of exposure. The daily dosage should not exceed 75 r. This amount of radiation usually causes little change in the leukocyte count but gradually causes a diminution in the size of the spleen and the patient's general condition improves. Usually, not more than five such treatments are necessary. Treatment is interrupted when the leukocyte count shows a tendency to decrease. For example, if the original count was only 5,000, treatment should be stopped when it has reached a level of approximately 4,000. If the count decreases after the first treatment, it is usually well to wait for from 24 to 48 hours before initiating the second treatment, because the count usually returns to the original level (or slightly higher), at which time treatment can be continued. If the original count was around 10,000, treatment can be continued until the count has reached a level of about 7,000. In treating patients who have leukemia of this type, we have seen remissions of the disease with almost normal health of the patient for as long as a year. Hematologic studies and palpation of the spleen should be carried out monthly.

When a course of treatment for a patient who has any type of myelogenous leukemia has been completed, a monthly leukocyte count should be taken and, provided the leukocytes do not tend to increase rapidly in number and immaturity, treatment should be withheld until such time as a definite increase manifests itself. Rather than to allow the leukocyte count to reach a hazardously high level, treatment should be resumed whenever a tendency to progressive increase becomes apparent. This procedure permits better control of the leukemia and the patient may continue to be a useful individual with the least amount of incapacitation. Moreover, less treatment is required to bring the leukemia under control.

The follow-up procedure for this condition is almost as important as the treatment itself. With such a varying clinical entity as myelogenous leukemia, as in the case of other types of lymphoblastoma, a statistical analysis of survival after treatment among patients suffering from this disease does not show a true picture of the value of roentgen therapy. It is the consensus, however, that, as a palliative measure, roentgen therapy surpasses all other methods of treatment.

The technics which we have outlined herein seem to be definitely superior to

other methods which we have tried. Three things should be avoided. First to be shunned is the not infrequent habit of some roentgenologists of giving "a little bit" of roentgenologic treatment to the spleen without a clear knowledge of the true status of the disease and without a definite plan of treatment. The second action to avoid is irradiation of the long bones of the average patient since, in our experience, such irradiation occasionally has produced a rapidly fatal outcome in cases in which the patients otherwise were progressing favorably. The third procedure to be avoided is the use of 200 kv., even in mild doses. In our experience this type of treatment may produce a more spectacular and more rapid immediate result than that which we have discussed, but the condition of patients so treated seems to become refractory much sooner than that of patients who have been treated with a technic similar to the one we have discussed.

SUMMARY AND CONCLUSIONS

This paper offers nothing new in the treatment of subacute, chronic, or aleukemic myelogenous leukemia, but it does offer a definite therapeutic plan which we have found to be very satisfactory. Treatment administered systematically, rather than sporadically, keeps patients in better general condition. We have also found that, by using a definite number of well defined roentgenologic fields at the time each course is given, the epidermis of the patient remains in much better condition and thus tolerates considerable repetition of treatment. We believe that by using a relatively small quantitative dose to each field of treatment, the leukocyte count decreases more slowly than it would otherwise and that the complete course can be administered without systemic reaction in almost every case. We have no evidence that stronger doses than those we have used produce better results in the average case.

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This point of view of the influence of the electric charge has been accepted by Fahraeus. Exposure to ultra-violet rays diminishes the potential of the electric charge of red cells and increases the rate of sedimentation, probably because it diminishes at the same time the repulse force between them, since they have always a negative electric charge. Likewise, theories have been established on the phenomenon we are studying, relating it to the index of blood calcium, to the CO_2, to the diminution in size of the red cells and their hemoglobin content, and to the alteration of the relation between albumen and globulins. Grandwohl attributes it to the *p*H, since acidosis delays and alkalosis increases the erythrocyte sedimentation rate.

Experiments of Maccabruni and Snaper tend to demonstrate that the corpuscles do not intervene in the sedimentation rate. If red cells of patients sick with some infectious disease are mixed with the serum of healthy individuals, sedimentation takes place normally. On the other hand, erythrocytes of healthy individuals mixed with diseased plasma, present an abnormality in sedimentation; from this these researchers deduce that sedimentation is influenced by the plasma and not by the corpuscles.

From what has been said, it is inferred that there are numerous factors influencing corpuscular sedimentation, and most probably there exist other causes still unknown.

The test is not specific, for which reason it has no diagnostic value, but, on the other hand, it does seem to have a great prognostic importance in some diseases. "The value of the reaction of corpuscular sedimentation is that of a general biological phenomenon, which always gives practically exact figures in normal individuals and may be raised in a passing or constant manner in the majority of acute or chronic diseases."

Cooper and Kürten mention acceleration in malignant tumors, believing this to be due to an increase in the amount of cholesterin in the blood. According to Forestier and Tonnet, it is very high in parenchymatous neoplasms, owing to phenomena of paradoxic albuminosis in the blood. Gram found a rise in the fibrin content of the plasma of nearly all infectious diseases, in carcinoma, etc.

The sedimentation test has come to be recommended as a help in differentiating benign from malignant tumors. It is generally admitted in the differential diagnosis of benign and malignant tumors that, under similar conditions, an increased sedimentation rate means malignancy. When not complicated by infection, fibromas, lipomas, and fibromyomas have no influence on sedimentation. However, in the earliest onset of a malignant tumor, the sedimentation rate may not be altered. It has been observed that there is no change in the phenomenon of Fahraeus in some cases of cancer of the cervix and of scirrhous carcinoma of the esophagus and stomach. Peters has pointed out that normal sedimentation figures are observed in epithelial cancer, as in that of the tongue and of the penis. Katz and Leffkowitz mention as possible causes affecting the stability of the formed elements, the anatomical character, its situation, vascularity, tendency to invade the neighboring tissues, degree of reabsorption of the tumor tissue, of the inflammatory reaction and the presence and state of metastasis. These authors think the reason why malignant tumors present modifications of sedimentation is because they show a tendency to disseminate.

I believe the study of the sedimentation rate to be of great interest in cancerous patients, following the evolution of the disease and relating it to irradiations. As pointed out, in the probable causes of sedimentation, many occur in patients with malignant neoplasms, either chemical modifications or biological elements constituting it. I ignore up to what degree irradiations modify the electric charge of the red cells, but if ultra-violet radiations are capable of altering it and producing acceleration of the sedimentation, it is

logical to suppose that x-rays in the form of deep therapy, as well as radium irradiation, have a more marked effect than ultra-violet rays. With such belief, I have irradiated guinea pigs and have invariably observed that the irradiation effect gives rise to an increase in the sedimentation rate. I chose these animals because they can be bled easily by puncturing the heart. At the same time, apart from the already well-known organic changes which irradiation causes when the animals survive to the irradiation, the sedimentation rate becomes normal, parallel with the descent of the irradiation curve. Consequently, I do not agree with the opinion somewhere expressed that irradiations do not affect the rate of sedimentation. I have measured the sedimentation rate in women suffering from uterine fibromas and found it normal before irradiation, and modified afterward, the same as happens with guinea pigs. The same thing occurs in cases of prostatic adenomas.

The cases studied are not in a sufficient number to allow me to present statistics and, still less, conclusions. The object of this paper has been to awaken among radiologists an interest in the study of a subject well worth while, in my opinion. Compared with other diseases, principally tuberculosis, I have found very few papers treating the modifications of the corpuscular sedimentation in cancer relating to irradiations, and in some of these papers contradictions are found. If investigations are carried out at the same time in different countries, following the same technic and a similar scheme in the observations, by the next International Congress we shall be in a position to confirm the observations made or to rectify them. As I have previously said, those that I have had the opportunity to follow make me foresee important possibilities in the study of the sedimentation rate with respect to the evolution of cancer in patients treated by irradiation.

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FATE OF CRANIAL DEFECTS SECONDARY TO FRACTURE AND SURGERY[1]

A FOLLOW-UP STUDY OF 150 PATIENTS

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STUDIES of the persistence of fracture lines have been much neglected in medical literature, though the scientific and medico-legal value is of considerable importance (Stewart, 1925; Glaser and Blaine, 1936). From a clinical viewpoint it is extremely valuable to be able to prognosticate the persistence of cranial defects, either traumatic or operative, and further, to be able to inform the patient of the length of time necessary for a bone graft to fill such defect.

An intensive and prolonged study shows that the healing of linear fractures depends, to a great degree, upon the age of the patient. The disappearance of the fracture line occurs much earlier in children than in adults. The width of the fracture also plays a part in the prolongation of its visibility upon the roentgenogram. Occasionally, if the fractures are too wide, absorption rather than healing of the fracture takes place. Ossification around the fracture and of the fracture itself may also occur. The location of a fracture in the skull also is an essential factor to be considered. In certain instances, fractures in the occipital area take much longer to disappear than in other parts of the cranium. It is well known that delayed union, and sometimes non-union, of fractures of the long bones occur: whether a similar bio-chemical process takes place in skull fractures is a matter yet to be ascertained.

In this series of 150 patients, x-ray films have been taken at varying intervals following fractures to determine the ability of the skull to repair such defects. In all instances, attention was directed to a duplication of the x-ray technic carried out in the original roentgenograms. The entire group has been classified as follows:

1. Linear Fractures:
 1. Disappearance of fracture line in children under six years of age.
 2. Disappearance of fracture line in adults.
 3. Widening of fracture line in children and adults.
2. Depressed Fractures:
 1. Without elevation of fragments.
 2. Hammering out of fragments.
 3. Cranial defects.
 4. Replacement of fragments at time of operation.
 5. Osteoperiosteal grafts.
3. Osteoplastic Bone Flap:
 1. Normal bone flap.
 2. Degeneration of the bone flap.

LINEAR FRACTURES

1. *Children.*—There are eight children under six years of age in this group. Fading of the fracture became apparent within two months after injury, and within six months after injury the fracture line disappeared in every instance, excepting one.

Case 1, S. F., male child, aged six years. This child was struck by an automobile, the wheels running over his head, giving him a tremendous fracture of the skull with many branches of the fracture line. There was considerable separation of one of the more extensive fractures, passing practically the whole length of the vault. The child was rendered unconscious for several hours, was irrational for several days, but after a stay of some three weeks in the hospital, made an uneventful recovery.

[1] Accepted for publication in September, 1939.



Fig. 1.



Fig. 2.

Fig. 1. Lateral view of skull showing wide separation of fracture line. Note the ramifications.
Fig. 2. Lateral view of same case taken nine years later. The skull has become irregular around the fracture line, and there has been a marked absorption in the center of the fracture. Parts of the linear fracture have, however, disappeared.

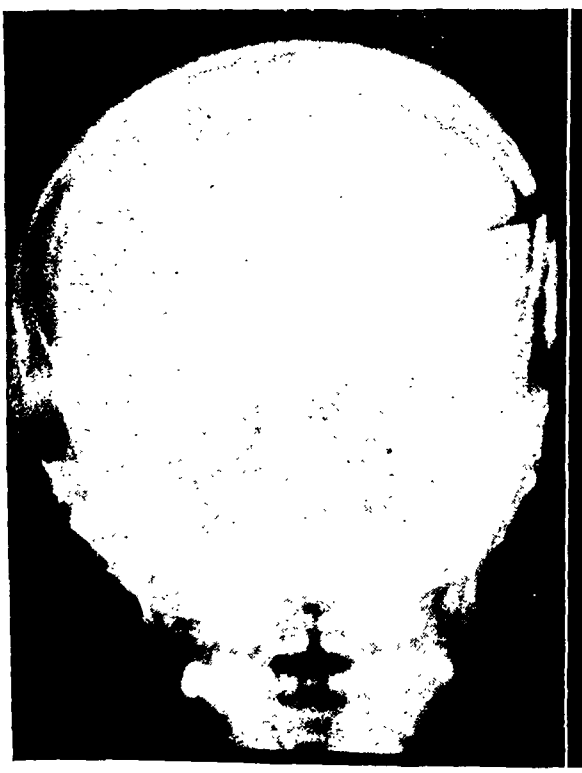


Fig. 3.

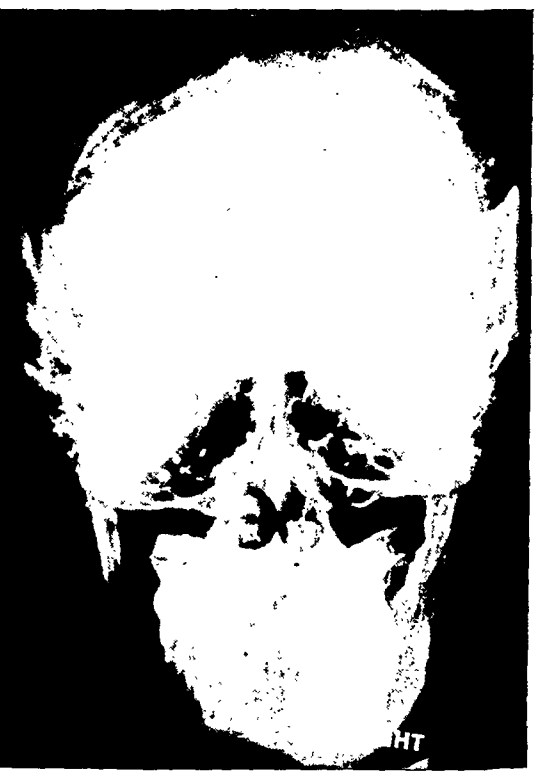


Fig. 4.

Fig. 3. Same patient as shown in Figure 1. The fracture is of a stellate type with a very wide separation.
Fig. 4. Same patient nine years later. All the fracture lines have disappeared except the widely separated area. This has become even wider due to absorption around the fracture line. The patient is now 14 years of age.

The extent of this fracture may be noted in Figures 1 and 3. X-ray films were made over varying intervals of time, and finally after a nine-year period the lateral film revealed a wider absorption of the center of the linear fracture. There was increased calcification of the bone above this fracture, with definite calcification along both margins of the transverse branch of the main lateral ramification. In the postero-anterior view it may be noted (Figs. 2 and 4) that most of the fracture lines have entirely disappeared. However, the main central fracture instead of disappearing has become definitely widened, with considerable absorption of the area superior to the crevice. In this instance, the continuation of the defect was due to the extensiveness and wide separation of the fracture. In addition, the increased calcification about the fracture line, and superior to it, was a rather unusual occurrence in a child of this age, this being the only case in our series which presented this picture. In the remaining patients, the fractures disappeared within the six-month period. Without doubt this fracture will remain visible during the entire life of the patient.

2. *Adults.*—In the 55 cases of adult linear fractures, the disappearance time was extremely variable. The earliest time for fading, so that the fracture was not distinguishable unless the original films were reviewed, occurred within seven months. It is quite apparent that those fractures which existed in the occipital region took a much longer time to disappear than those appearing in other parts of the skull. In this group, some fractures of the occipital region were visible for an eight-year period. In four examples, instead of a healing, a widening of the fracture with absorption about the edges occurred. In all these cases, the separation was rather extensive, though in some, a wide separation did not follow such a course. The following five cases present examples of the varying kinds of fading, from a seven-month period to an eight-year period, and also give an example of absorption about the fracture.

Case 2, R. S., aged 40, a frontal parietal fracture faded to practical invisibility after seven months.

This man had a severe head injury on Sept. 1, 1935, when he was rendered unconscious, but did not have a skull fracture clinically or roentgenologically. Following this injury he developed a disabling post-concussion syndrome, associated with headaches and dizziness, as well as marked

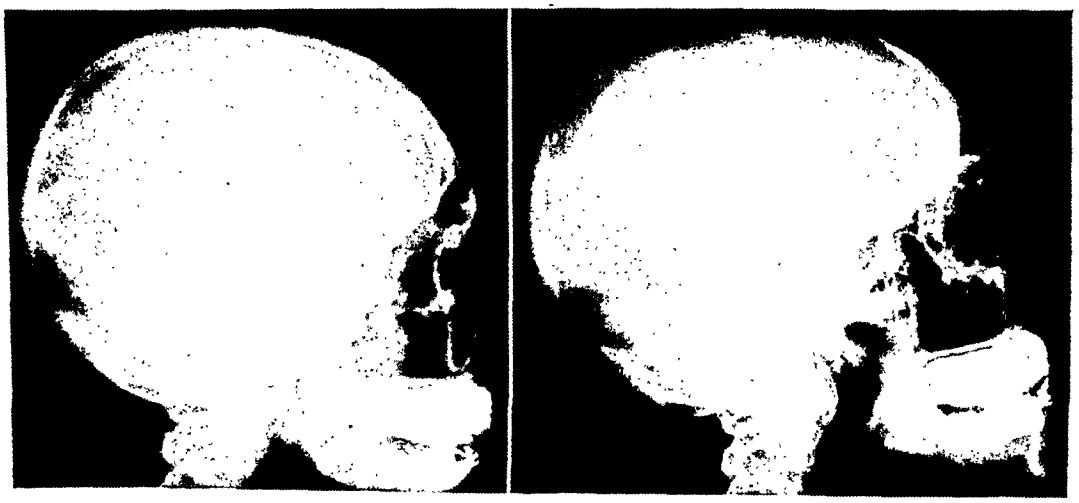


Fig. 5. Fig. 6.

Fig. 5. Linear fracture in adult aged 40 is clearly visible in the frontal region.
Fig. 6. The same linear fracture has practically disappeared within seven months. Without the original films the existence of this fracture could not be definitely ascertained.

nervousness. At a later period, he developed a post-traumatic epilepsy. During one of these epileptic seizures, on Dec. 15, 1938, he fell to the ground. This time he did receive a skull fracture and was again rendered unconscious (Fig. 5).

His headaches and dizziness became more severe, and he tired more readily. His vision became blurred and his unconscious attacks increased in frequency so that he had as many as 30 attacks in one year. Physical examination revealed a well built, sturdy individual. His septum was deviated slightly to the right. There was a polyp in his right nostril, and his teeth were in relatively poor hygienic condition, with partial upper and lower plates. In addition, he had a moderate kyphosis. Neurologic examination revealed some tenderness in the right occipital region. There was an irregularity in his pupils, and he reacted sluggishly to light and distance. His reflexes were within normal limits, but there was a marked tremor of his eyelids. He had a tendency to sway while standing with his eyes closed, although this latter was not a true Romberg. His blood and urine were normal. Wassermann test was negative. X-ray examination, repeated on June 2, 1939, some seven months after the original injury, revealed a marked fading of this fracture, although a definite diagnosis could not be arrived at unless the original films were reviewed (Fig. 6).

Case 3, J. G., aged 34, weight 144 pounds, height 5 feet 3 inches. A large fronto-parietal fracture, fading to near invisibility 10 months after injury, and to absolute invisibility in 14 months after injury.

This man had a severe head injury on April 20, 1938, falling a distance of 12 feet to a concrete floor below. He was immediately rendered unconscious and remained so for one-half hour. He had no memory of events during the first 14 hours. In addition to his skull fracture,



Fig. 7.



Fig. 8.

Fig. 7. J. G., aged 34, received extensive frontoparietal temporal fracture. There was considerable separation in the frontal region.

Fig. 8. Film of the same patient, taken 10 months later. Note the gradual fading of the fracture line.

he had a fracture of the left radius and ulna at the wrist and developed a cerebral spinal rhinorrhea, as well as an associated diabetes insipidus, drinking between two and three gallons of water a day. His right eye was closed and swollen and he had numerous body abrasions and contusions, but his neurologic examination was essentially negative. After he regained consciousness, he complained of headache, but was entirely without dizziness. He remained in the hospital until May 15, then rested at home for an additional three weeks. He had few complaints and gradually returned to his occupation as storekeeper. His diabetes insipidus has persisted to the present time, though now there is only a slight excess in his water-drinking.

X-ray examination on April 21, 1938 (Fig. 7), showed a fracture in the right frontal region, about three inches in length, extending through the frontal sinus and involving both the anterior and posterior walls. The entire sinus was clouded and filled with blood. Another fracture was visible in the parietal region on the right side, about five inches in length, entering the mastoid area, which also was somewhat cloudy. X-ray film taken on Feb. 6, 1939, 10 months after injury (Fig. 8), revealed a remarkable fading of the fracture line, far more than would ordinarily be expected. The rather wide frontal fracture was narrowed and the parietal bone fracture was barely visible. X-ray films taken on June 29, 1939, 14 months later, revealed a nearly complete disappearance of the fracture line. The only indication discernible was the lower border of the parietal line where it had entered the mastoid. This in itself would not be recognized as a fracture, unless the previous films were used in comparison.

Case 4, A. S., aged 23, weight 170 pounds, height 6 feet one-half inch. An extensive fracture extending over the entire lateral half of the skull showed marked fading, though readily distinguishable upon x-ray examination, 17 months after injury.

On July 5, 1937, this young man was struck on the head by a timber from an oil derrick, and was rendered unconscious for a lengthy period of time. He has no recollection of events for a period of two weeks after the injury. He remained in the hospital seven weeks, and then reported for office examination. On Dec. 7, 1938, he still complained rather severely of headaches and dizziness, weakness, fatigue, irritability, and back pain. After

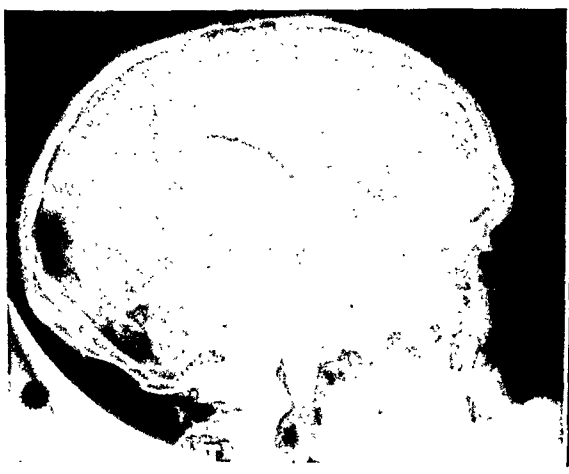


Fig. 9.

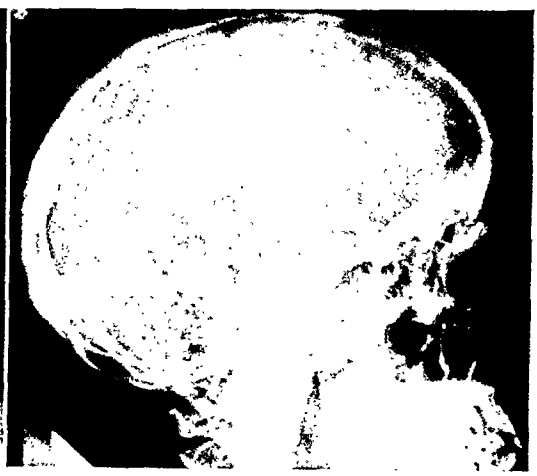


Fig. 10.

Fig. 9. A. S., male, aged 23. There is extensive fracture with wide separation over the lateral side of the skull.

Fig. 10. Same patient, 18 months later, still showing evidence of the fracture present. However, marked fading had occurred.

Additional fractures were seen running in different directions in the left parietal bone, also involving the squamous portion of the left temporal bone. Figure 14, taken one year later, showed very little change. In it, some depression is seen at the vertex. The anterior fragment has settled down a distance equal to the complete thickness of the skull wall.

DEPRESSED FRACTURES

There were 79 individual cases of depressed fractures. In the group without operative elevation, the fragments fused together to form a solid rounded posterior edge, as well as a similar rounded anterior edge in every instance. The time of fusion varied from 10 to 14 months. In one instance, a bone flap was turned down and the fragment hammered in place. With the bone flap replaced, no evidence of the fracture was visible after a 10 months' period. This operation was too formidable, and is deemed unwise for the treatment of depressed fractures. In those cases in which the fragments were removed and a defect in the skull remained, this defect never decreased in size. The only change noticeable was a rounding of the serrated edges within an eight months' period of time. This occurred in ages from six to sixty years. In certain instances the fragments were replaced. If spaces were left between the replaced fragments and the edges were not approximated, or bone dust not inserted between these edges, the x-ray evidence of separation is maintained permanently, up to five years. There is no indication of fill-in, yet, to palpation, the surface appeared as if solid bone were present. In order to maintain the position of the fragments in close approximation, a catgut screen has been devised. This assures that the smaller pieces of bone will not become misplaced, and the scalp may be firmly sewed together. In such instances asepsis must be assured and the dura must be intact. Graft or suture is necessary in all such cases. Experimental studies by Glaser and Thienes demonstrate that an animal membrane graft is just as efficient as fascia lata. It has been found

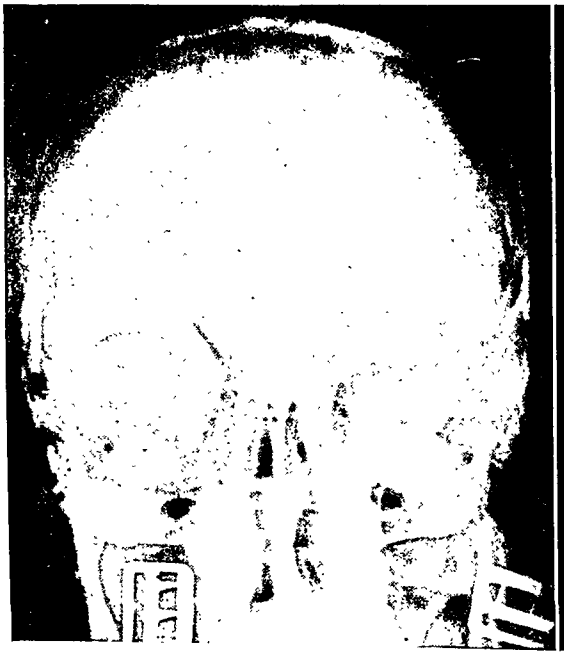


Fig. 15.

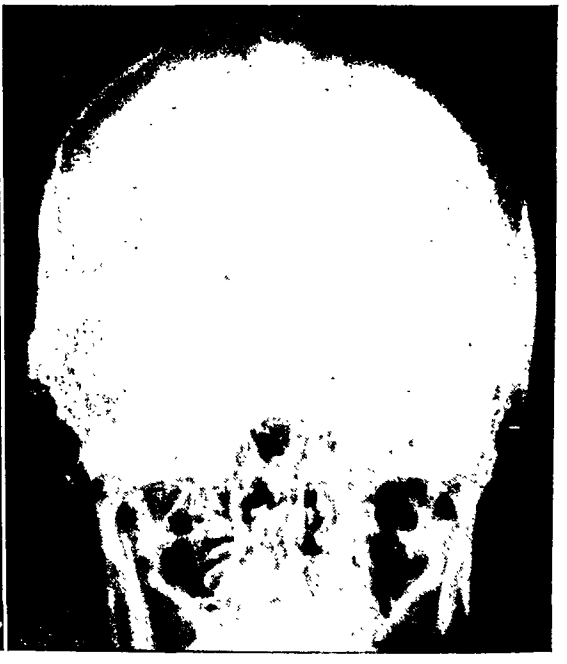


Fig. 16.

Fig. 15. A depressed fracture in the temporal region which has not been elevated. Note the fragmentation of the depressed bone.

Fig. 16. A film taken 23 months later showed a fusion of the fragmented particles, with a rounding and smoothing of the dural edges.

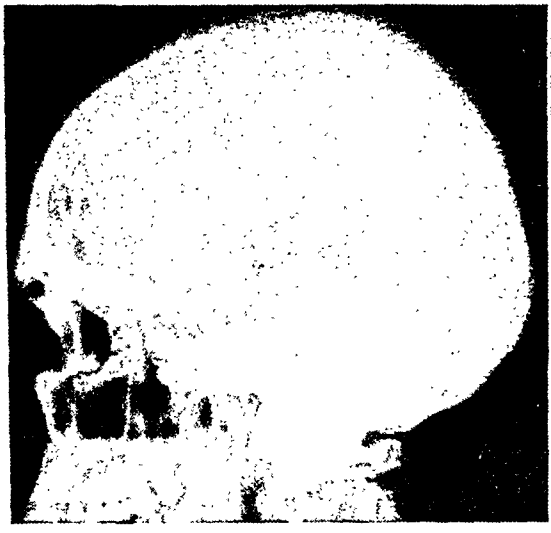


Fig. 17.



Fig. 18.

Fig. 17. A depressed fracture with elevation and removal of the fragments. Note the fine serrated edge.
Fig. 18. X-ray films taken eight months later revealed the smoothing of this serration, without any decrease in size of the defect. This defect never became smaller.

that osteoperiosteal grafts are the most satisfactory, both from a technical and a clinical standpoint (Glaser and Shafer).

Case 7, F. S., aged 57, weight 175 pounds, height 5 feet 10 inches, received a depressed fracture which was not elevated. X-ray films taken 23 months later showed a fusion of the fragments, with a rounding and smoothing of both edges.

This man fell a distance of 12 feet, striking the ground, being rendered unconscious for six minutes. Upon examination he was found to be entirely symptom-free, except that he was somewhat drowsy. He had a blood pressure of 165/105, and a definite auricular fibrillation. The hygienic condition of his mouth was extremely poor; tonsils large and infected. In addition, he had a fracture of the clavicle and two metacarpals of the left hand. Neurologic examination revealed an edematous and discolored face, rupture of the left tympanic membrane with hemorrhage, tenderness, and swelling over the temporal region of the scalp. Due to this man's auricular fibrillation and a complete absence of neurologic signs of a focal or generalized nature, elevation was deferred. He was discharged from the hospital at the end of four weeks and returned to his usual labor six weeks after injury entirely symptom-free. The auricular fibrillation persisted, and because of the absence of any head symptoms, elevation was never carried out. He died seven years later from heart disease.

X-ray examination of the skull on May 22, 1931, showed a depressed comminuted fracture of the left parietal bone. The



Fig. 19. The fragments have been replaced in a depressed fracture. However, the bone has not been closely approximated. This spacing has not been covered by bone over a period of four years.

fragment was somewhat quadrilateral in shape and wider anteriorly. In the anterior portion it measured 1¾ inches, whereas in the posterior portion it measured 1 inch. The upper fracture extended in almost a straight line from the coronal suture to the lambdoid suture. The lower fracture extended practically the same way with the exception that there was an overriding, especially in its posterior aspect. Another fracture was visible in the inner table about the center of the fragment (Fig. 15). Examination of the skull on April 12, 1933, revealed rounding and fusion of the fragments into one solid mass with even some filling-in of the surface depression (Fig. 16).

As has been previously pointed out by Glaser and Shafer, the early elevation of depressed fractures is not essential in all instances. In particular cases elevation may be entirely eliminated without any danger to the patient whatsoever. The following case represents such an example.

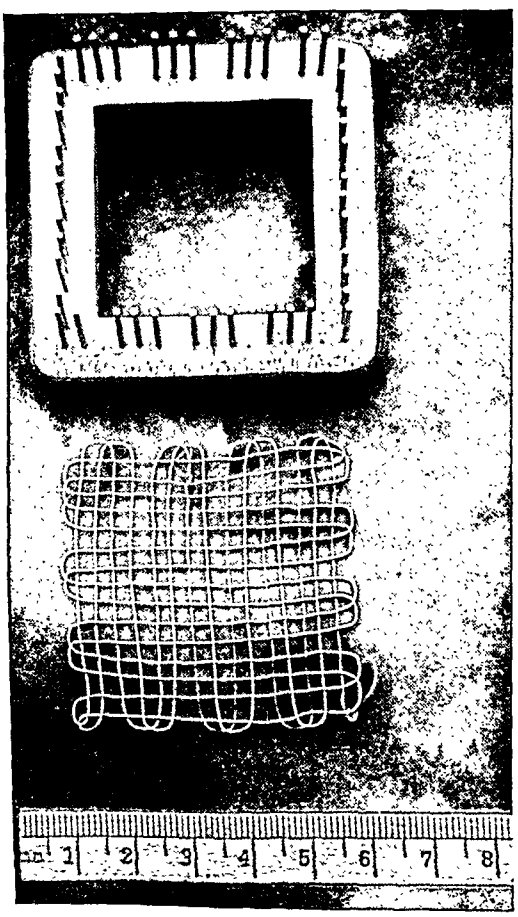


Fig. 20. In order to facilitate surgery a catgut screen has been devised to hold the fragments in place, and also to aid in maintaining position of very small fragments so that complete bony union may occur.

Case 8, G. L., male, aged 25 years, weight 165 pounds, height 5 feet 11 inches, was struck by an automobile, this injury resulting in a dirty, contused, and lacerated scalp, with a deep depression of the skull and a rupture of the dura. Due to the possibility of infection occurring in the wound, it was deemed advisable to remove all fragments of bone. The defect did not decrease in size, and eight months later the serrated edges became smooth.

This patient was injured on May 5, 1936, being rendered unconscious for 30 minutes. He was immediately removed to the hospital. Physical examination was essentially negative, except for abrasions and contusions about the back and shoulder. Neurologic examination revealed a ragged, crisscross, dirty wound in the right temporal region, oozing considerable blood. The examination was entirely negative, save that the patient proved somewhat sluggish in responses. Several x-ray film, taken on May 5, 1936, show a rounded depressed fracture in the right temporal region with the center fragments depressed three-quarters of an inch beneath the surface. These fragments were all removed at operation. X-ray examination one week later revealed a bony defect (Fig. 17), with very slight serration of the edges. X-ray examination eight months later demonstrated a defect of the same size, the only change noticeable being the rounding of the serrated edges (Fig. 18).

It has been the contention of some that the aperture decreases in size, particularly in children. In this series, the youngest patient was six years of age. In this case, x-ray examination eight years later revealed no decrease whatsoever in size. In the case of adults some of these apertures were present as long as thirty years. In carefully checking the size of the apertures, it is well to measure the open-

ing by calipers. In all instances no decrease in size occurred and the only change noticeable was the rounding of the edges, as demonstrated in this case.

Case 9, E. J., male, aged 31 years, weight 175 pounds, height 5 feet 10 inches, was struck on the head by a falling derrick. The primary wound was sutured. Three weeks later the depression was elevated, and the fragments placed *in situ*.

This man was rendered unconscious for a short period of time. He regained consciousness in the ambulance on the way to the hospital. After his entry into the hospital, he again lapsed into unconsciousness and remained so for several hours. Physical examination revealed a rupture of the right tympanic membrane, with bleeding from the right ear. Neurologic examination revealed a lacerated wound in the posterior parietal region, and a depression in the underlying skull. There was some loss of hearing in his right ear. He was in shock. No evidence of intracranial pressure, or focal signs of hemorrhage were shown. He developed a mild otitis media, but the laceration in his scalp healed *per primum*. Three weeks later surgery was performed and the fragments placed *in situ*. The patient returned to work, symptom-free, four months after injury.

Figure 19 demonstrates the position of the fragments one year after injury. There has been no filling-in of the space between the fragments other than the posterior area wherein a certain amount of overlap existed. This region was quite firm to palpation and felt as if the bone was completely united. Films taken in similar cases up to four years after replacement showed a similar spacing.

Realizing the difficulties in placing small pieces of bone in position and maintaining them so that proper healing of the defect may occur, a catgut screen made of woven catgut, as indicated in Figure 20, has been constructed. This screen is sterilized and sewed *in situ*, thus acting as a support for maintaining the position of the fragments and bone dust. By this method, a complete closing of the defect may be obtained. Figure 21 demonstrates close approximation of practically all the fragments in a depressed fracture situated in the frontal region, taken 14 months after injury.

Case 10, W. H., male, aged 51 years, fell 20 feet into a mine shaft. Osteoperiosteal graft was inserted at a later date. Three and a half years after insertion there was only a small defect visible.

This man was dazed, but not unconscious. He had a large laceration in the left occipital region, with a depression of the bone. The case was examined two days after injury and at that time there were no abnormal, neurologic, or physical findings. Due to the fact that the wound was potentially infected and a great amount of débris was situated beneath the scalp, it was deemed good surgical judgment to remove the fragments. He made an uneventful recovery and returned to work three months after injury.

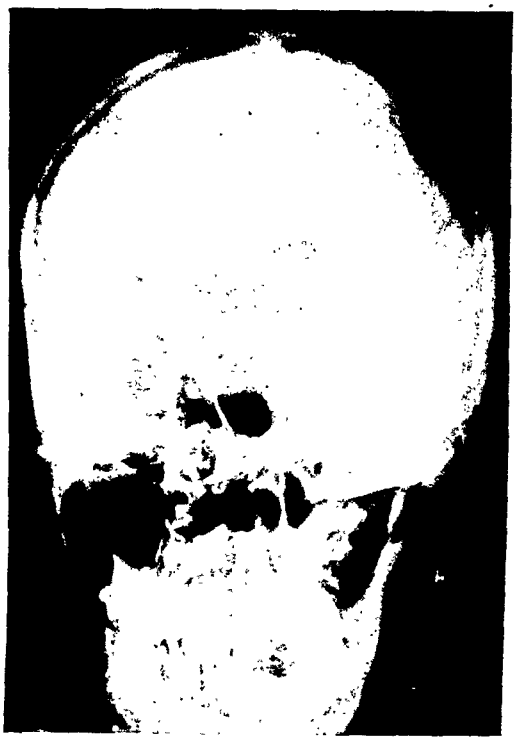


Fig. 21. Closely approximated fragments in the frontal region (right) have united firmly to close the defect almost entirely.

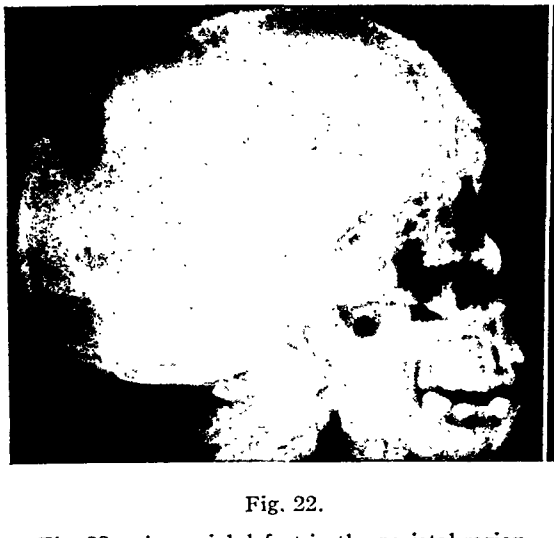


Fig. 22.

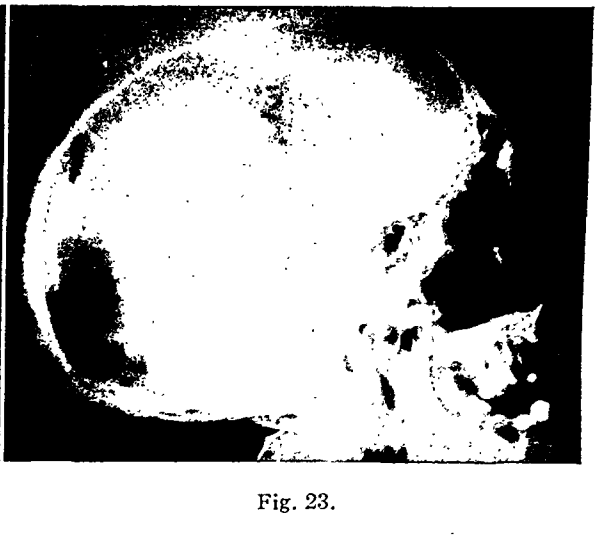


Fig. 23.

Fig. 22. A cranial defect in the parietal region.
Fig. 23. The same patient after an osteoperiosteal graft had been inserted. This film, made three and a half years later, demonstrates the decrease in size of the defect. Larger grafts have taken as long as five years to fill in to this extent.

Later a mild skin infection necessitated treatment of the wound. Due to the mildness of the infection, operative procedure was postponed for a long period of time. On July 2, 1934, the wound was prepared for surgery, but a small pocket of pus was found within the scar. On May 30, 1935, he was again prepared for surgery. X-ray examination at the time of removal of the fragments and in May, 1935, had revealed a cranial defect in the occipital region (Fig. 22). An osteoperiosteal graft was inserted; the patient made an uneventful recovery. X-ray film taken three and a half years later showed a definite decrease in the size of the bony defect (Fig. 23).

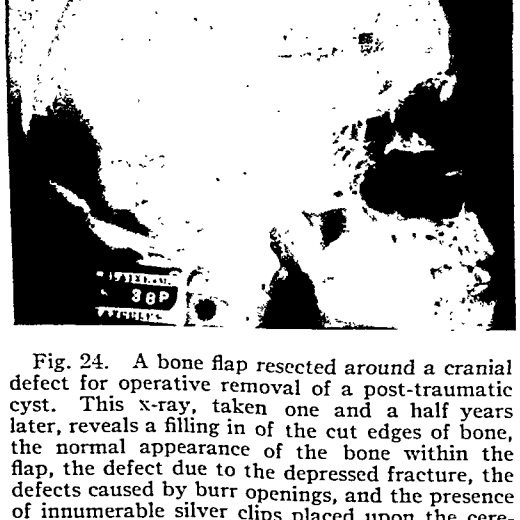


Fig. 24. A bone flap resected around a cranial defect for operative removal of a post-traumatic cyst. This x-ray, taken one and a half years later, reveals a filling in of the cut edges of bone, the normal appearance of the bone within the flap, the defect due to the depressed fracture, the defects caused by burr openings, and the presence of innumerable silver clips placed upon the cerebral vessels when the cyst was removed.

It has been our experience that the use of the osteoplastic graft gives the best results for the closure of the aperture. The introduction of silver plates is extremely dangerous. Infections, or the falling out of the plate, even after long periods, may occur after the procedure. In all of our cases we have been fortunate to have the grafts take, and the apertures gradually become smaller. At all times the graft feels solid to touch and acts as a protective covering to the underlying brain. The good results obtained with these osteoperiosteal grafts taken from the skull, and the simplicity of this procedure, obviates any need to go to other parts of the body for bone.

OSTEOPLASTIC BONE FLAPS

Osteoplastic bone flaps turned down for the removal of brain tumors, hemorrhage,

or other intracranial pathology undergo various types of changes. In infants, before complete ossification of the bones occurs the wounds will heal completely without signs of operative procedure. In adults, the bone flap itself may appear quite normal, or in certain instances calcification within the center of the flap occurs. The edges of the flap and the burr holes may even disappear to a great degree, if close approximation of the flap is carried out and if bone dust is placed in the burr opening. The following two cases demonstrate this.

Case 11, A. S., male, aged 24 years, suffered a depressed fracture from which he developed a post-traumatic epilepsy, secondary to a brain cyst. At a later date a bone flap was turned down to remove the cyst. The bone flap appeared normal two years after injury.

On Dec. 4, 1933, the patient was struck by an automobile and rendered unconscious. He suffered a very severe depressed fracture of the skull, the scalp was lacerated, and the fragments of bone were both deeply embedded in the brain, as well as loosely in the scalp. The dura was badly lacerated and considerable brain tissue oozed from the wound. Due to his precarious condition, emergency measures were instituted, consisting of the removal of the bone fragments and suture of the scalp. He remained unconscious for weeks and hovered between life and death for the first two weeks, yet he eventually recovered. On July 17, 1936, he developed his first convulsion. These convulsions were not of a focal nature. Encephalograms revealed the presence of a brain cyst, and, on June 26, 1937, a bone flap was turned down, and the cyst was resected. Since then he has been entirely free from convulsions. X-ray examination two years later revealed the bone flap in good position, the burr holes quite distinct, and in the posterior edge the healing of the flap to the skull was seen to occur. The central bone defect did not decrease in size, but did not interfere with the circulation of the flap. On the x-ray film (Fig. 24) the numerous silver clips placed in the brain for hemostases may also be noted.

Case 12, E. P., male, aged 28 years, developed a subdural hematoma which was immediately removed. The bone flap, turned down to permit removal of this hemorrhage, degenerated over a period of four years.

This patient had been struck on the head in a hold-up. He was immediately ren-

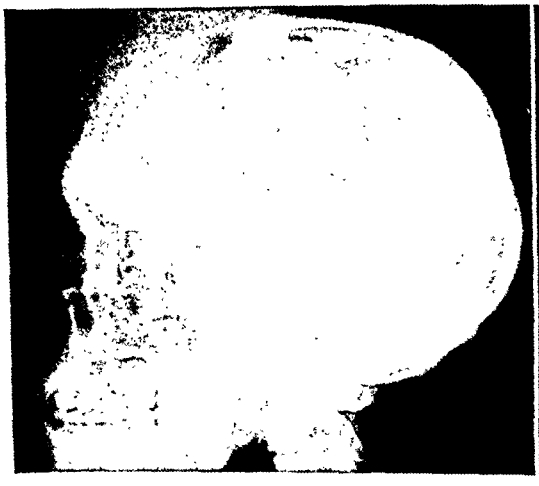


Fig. 25.



Fig. 26.

Fig. 25. Film taken one and a half years after an osteoplastic flap had been turned down. Note beginning changes about edges of flap and in the center.

Fig. 26. Film taken six years later revealed a marked decalcification in the center of the flap. No further changes had occurred in the six-year period.

dered unconscious and remained so for several hours. Within a three weeks' period, focal signs, as well as evidence of increased intracranial pressure, developed. A diagnosis of a subdural hematoma was made, which was removed by the turning down of an osteoplastic flap. The patient made an uneventful recovery, but one year and a half later developed a generalized epilepsy. X-ray examination at that time (Fig. 25) showed the bone flap *in situ*. In Figure 26, made four years later, there was a generalized decalcification of the bone flap. To palpation, however, this flap was firm to touch. Encephalograms taken upon this patient revealed markedly distorted and irregular ventricles, with an entire shift of the ventricular system. Under barbiturates he has been entirely free from convulsions.

CONCLUSIONS

I. Linear fractures in children under six years of age disappear within six to twelve months after injury, except in instances in which the separation of a fracture has been extremely wide. In those cases the defect may persist permanently.

II. Linear fractures in adults situated in the frontal, parietal, and temporal regions begin to fade immediately. However, complete disappearance rarely occurs under seven months, and in these cases comparison with the original film shows evidence of fracture. The average time of complete disappearance of these fractures, even though fading is evident upon the x-ray film, is three to four years.

III. Fractures in the occipital region take much longer to disappear, and in this series evidence of fracture has persisted as long as eight years, though fading is apparent.

IV. In some instances the area about the fracture absorbs, rather than disappears, leaving a much larger x-ray shadow defect than at the time of injury. This may occur in children as well as adults. The result is a permanent defect.

V. In depressed fractures without elevation, the fragments become rounded and unite, and the lines of fracture cannot be detected, though the depression is apparent.

VI. In operative defects, wherein the bone has been removed, or in cases of depressed fracture wherein the fragments have been removed, the cranial defect never becomes smaller, the only change being a rounding of the edges.

VII. When a bone flap has been turned down around a depressed fracture, and the depressed area carefully hammered out, no evidence of depression or fracture lines will appear upon x-ray examination.

VIII. Bone fragments placed *in situ* at the time of an operation for depressed fracture must be closely approximated, so that all the edges touch, in order to secure complete bony union; otherwise the spaces between the fragments will be visible upon x-ray examination.

IX. Osteoperiosteal grafts are the best means of covering skull defects. They remain firm and solid to the touch and complete calcification depends upon the size of the defect. It requires from five to eight years before the opening of average size is completely calcified.

X. Bone flaps may either undergo absorption, or appear normal.

XI. X-ray films taken long periods after skull injury are just as important in determining the existence of fractures in medico-legal cases as those taken immediately. By a careful study of these films the approximate age of the fracture may be determined in many instances.

DUODENAL ULCER IN THE PRESENCE OF A GALL-BLADDER SYMPTOMATOLOGY

By ERIC J. RYAN, M.D., St. Luke's Hospital, *New York City*

IN its typical form, peptic ulcer gives a symptom complex which runs more true to form than is the case in almost any other condition. The characteristics for peptic ulcer in general may be roughly divided into four as follows: First, meal relation; second, food ease; third, intermittance, and fourth, chronicity.

There is a variation in the symptomatology, particularly as regards food ease, between ulcers in the prepyloric region and those in the postpyloric region. Moynihan (1) has aptly illustrated this in the sequence of pain in relationship to food, in the following manner:

> "In cases of gastric ulcer, the pain which, after an interval, follows the taking of a meal, gradually disappears before the next meal. In cases of duodenal ulcer, the pain continues until the next meal, or until food is taken to give ease to a wearisome pain. The rhythm of gastric ulcer is 'food, comfort, pain, comfort': of duodenal ulcer it is 'food, comfort, pain': a quadruple rhythm in the former disease, a triple rhythm in the latter."

Following the ingestion of food, pain is liable to occur at an earlier time in gastric ulcer than in duodenal ulcer. A characteristic which is frequently noted is the occurrence of night pain, which may come on at the same hour every night. The type of food ingested may cause greater or less pain, the aromatic vegetables and heavy meats producing the most severe type of pain. In duodenal ulcer, particularly, although these same foods may give relief more rapidly, pain tends to return in a more severe form.

In both gastric and duodenal ulceration, a certain number of individuals will give a history which is in no way typical of either condition. In going over a group of cases operated upon at St. Luke's Hospital, New York City, for gall-bladder disease, I was interested in the number in which either mild or moderately severe cholecystitis was noted, with adhesions to the duodenum and the presence of single or multiple ulcers in the adherent area. The interesting question that arises is, which came first, the cholecystitis or the duodenal pathology?

Among many theories as to the cause of peptic ulcer (Nelson, 2), there is mentioned an anatomic relationship between the gall bladder, appendix, and the stomach and duodenum through the related vascular supply of the omentum. The causes of peptic ulcer are generally considered to be thermal, chemical, bacterial, or nervous. Whatever the etiologic factors may be, it is generally accepted that the final stages are (*a*) lowering the resistance of the intestinal mucosa, possibly by altering the production of their protective secretions, and (*b*) allowing digestion of the mucosa by the acid pepsin formed in its own glands.

Ivy and Fauley (3) draw attention to the rôle played by pylorospasm, which, in causing rupture of blood vessels in the stomach and duodenum, may give rise to ulceration. Gastritis and duodenitis may be the forerunners of chronic ulceration.

Among the cases previously mentioned, seven were of particular interest. In them there were certain points which would indicate the possibility that the lesion in the duodenum was secondary to pathology in the gall bladder. In these cases stones were found in the gall bladder, and a rather marked degree of cholecystitis was noted. In four of the cases a moderate degree of cholecystitis was noted with no evidence of stone in either the gall bladder or cystic duct. The absence of stones in the gall bladder with a mild degree of cholecystitis suggests (and the suggestion is borne out by the experience of several surgeons with whom I have had personal communications, 4) that the original pathology in these cases was in

[1] Presented before the Twenty-fourth Annual Meeting of the Radiological Society of North America, at Pittsburgh, Nov. 28–Dec. 2, 1938.

the duodenum, and by surrounding inflammation and the formation of adhesions had spread to the gall bladder. Case histories in two of these cases and a summary of the findings in the other five illustrate the difficulty in arriving at a correct pre-operative diagnosis in cases of this type.

Case 1. E. T., white female, 37 years of age, whose chief complaint was a pain in the abdomen. Regarding her present illness, the patient states that about 12 years ago, during pregnancy, she had an attack, diagnosed as gallstone colic. She suffered several attacks in the next three months and since then has had several a year. The attacks would come on suddenly and be very severe, the pain radiating to the left side, through the back, and up to the right shoulder. She has had considerable gaseous eructations, but her attacks had no apparent regular relationship to food. The last one left a residual soreness which is still present. She has had no jaundice and no evidence of blood in the stool. Both personal and family histories were negative.

On physical examination the abdomen was tender to palpation in the right upper quadrant; no rigidity nor masses were felt. Other systems were essentially negative. Pre-operative diagnosis was that of chronic cholecystitis and cholelithiasis. On operation the gall bladder was found to be bound with adhesions to the duodenum and showed considerable thickening and contained small stones. The duodenum in its gall-bladder aspect was found to be edematous and the wall indurated near the pylorus. On palpation an ulcer about one centimeter in diameter was found. On opening the duodenum, a kissing ulcer was found on the posterior surface.

Case 2. J. F. white male, 35 years of age, whose chief complaint was epigastric pains after meals for two or three months. About three months previously he began to have pressing, aching pains in the mid-epigastrium, coming on immediately after a heavy meal or about 1 A.M., after a heavy dinner at 7 P.M. The pains occasionally radiated to the left costal margins or up behind the sternum. Sodium bicarbonate (1 teaspoonful in half a glass of water) would be followed by relief from the mild pains in 20 minutes; more severe pains would improve only after vomiting. Attacks occurred more often, he observed, after eating heavy greasy food or soup. Six weeks previously after being wakened every night for a week at 1 A.M. with pain and vomiting, he began eating only light meals. He has been better since, and has had no pain for a week. He was not jaundiced. The stools were not black nor bloody, but light yellow. Personal and family histories were negative.

Physical examination revealed the abdomen to be symmetrical and relaxed. There was slight tenderness in the mid-epigastrium about one inch below the xiphoid, and in the right upper quadrant, beneath the costal margin about one and one-half inches lateral to the midline. There was no spasm, no masses felt. Other systems were negative. An examination of the gall-bladder region showed the gall bladder to fill with the dye. It showed poor emptying in response to food. There were no stones seen. Pre-operative diagnosis was chronic cholecystitis.

Surgical findings revealed the gall bladder to be the site of some adhesions. The wall had much the appearance of chronic cholecystitis; a good many adhesions to the gall-bladder wall both from the duodenum and transverse colon were seen. There were no stones in the gall bladder or common duct. Just beyond the pylorus on the antero-superior surface of the duodenum was a large penetrating ulcer, the surface of which was covered with lymph. The crater of the ulcer measured about three centimeters, and was rather deep.

It will be noted that in the first case the attacks were acute, unexpected, and gave no food relationship. The pain tended to radiate to the back; and whereas radiation of pain in this direction may occur in the presence of peptic ulcer, it is not typical. Typical pain tends to radiate anteriorly, particularly toward the sternum. Of the seven cases mentioned, three showed radia-

tion of pain posteriorly. In the others the pain tended to radiate to the left and upward under the sternum. In none of the cases was there continued food relation, although in two instances it was claimed that pain which followed the evening meal was relieved by the use of sodium bicarbonate.

The duration of symptoms in these patients ranged in order from three months to five years, and from eight to twenty-five years. All of them had periods of relief when no symptoms existed. All showed some of the features of ulceration, but none of them gave the typical complete syndrome. The complaints were such that in only two cases did the clinician feel that an examination of the gastro-intestinal tract was indicated. All had been referred to the X-ray Department for an examination of the gall bladder for pathology. In only one case was the gall bladder visualized. The others showed no filling, and the two cases referred for examination of the gastro-intestinal tract showed duodenal deformity.

An inflammatory process in the abdomen can, and frequently does, give rise to spasm of the sphincters, such as the pylorus, as is seen in the presence of a normal stomach and duodenum with a six-hour gastric retention; which spasm later is shown to be due to disease in either the gall bladder or the appendix. The anatomic relationship, through the circulation and lymphatic distribution between the gall bladder, stomach, and duodenum, is accentuated by the spread of an inflammatory process and the formation of adhesions between them, through which bacterial emboli may be carried in either direction.

An inflammatory process primary in the gall bladder may, by extension to the duodenum and by the spasm set up in the pyloric region, so alter the blood supply and function of the duodenum that an ulcer readily forms. An ulcer primary in the duodenum may likewise, by extension of its inflammatory process to the gall bladder, create a cholecystitis with or without the presence of stones.

It is interesting to note that in the presence of cholecystitis from either cause, the character of the pain, its usual direction, and the general symptomatology of the patient tend to overshadow the typical picture so frequently seen in the presence of gastric or duodenal ulceration.

Conclusions.—An inflammatory process may extend regionally from one organ to another and give rise to a secondary lesion in an adjacent organ; and the clinical picture may be so overshadowed by the symptoms arising from the secondary lesion that the primary lesion will remain clinically unsuspected.

Cases presenting a part, but not all, of the chief characteristics of peptic ulcer, and suggestive of gall-bladder disease, should be examined not only for a gall-bladder lesion, but should be followed by a complete examination of the gastro-intestinal tract.

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DISCUSSION

NATHAN B. NEWCOMER, M.D. (Denver, Colo.): In regard to the frequency of gall-bladder pathology and peptic ulcer in the same individual, I would like to quote from an article by Good and Kirklin, published in the *Collected Papers of the Mayo Clinic,* of 1936. In an analysis of 100 cases of peptic ulcers, they found 80 normally functioning gall bladders. In addition, there were eight normally functioning gall bladders in which they were unable to rule out stones; two normally functioning gall bladders with tumors; three normally functioning gall bladders with stones; two poorly functioning gall bladders; one poorly functioning gall bladder with stones; two non-func-

years, in giving the larger doses of dye, or the double dose of dye, if you choose to, our cases of lessened density in peptic ulcers have been markedly diminished and we now obtain better densities of gall-bladder shadows. However, I wish to emphasize again that in cases in which we do not obtain a good gall-bladder shadow, a large percentage of these show an associated gall-bladder pathology.

It is interesting, too, that even by the intravenous method, as Lieb has pointed out, 26 per cent of organically normal gall bladders failed to fill in 100 cases of duodenal ulcer. This figure seemed to be rather high.

I am inclined to believe that dyskinesia of the gall bladder is due to its close proximity to the duodenum, causing associated symptoms as a result of cholecystic duodenal adhesions or excessive irritability.

ERIC J. RYAN, M.D. (*closing*): The subject under discussion interests me because it is, as I previously said, an incomplete examination, and for accurate preoperative diagnoses, we have to convince the clinicians of the necessity of a complete examination.

We frequently encounter these cases. In my abstract I have changed, a little, the intent of my paper; I did so purposely to give rise to discussion. I think when we have indefinite findings in cases of this type we should insist more and more on as complete an examination as possible.

MILLIAMMETER INACCURACY CAUSED BY CONTROL CABINET[1]

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From the Division of Physics and Biophysical Research, the Mayo Foundation

WHILE calibrating an x-ray therapy machine it was noticed that the milliammeter in the aerial system read about 10 per cent higher than the milliammeter on the control cabinet which was in the grounded circuit of the secondary of the high voltage transformer. The aerial milliammeter was checked with a laboratory meter, battery, and rheostat, and found to furnish correct readings to within ± 1 per cent. The milliammeter in the control cabinet was checked with the same laboratory meter, both with the battery and while the x-ray machine was in operation, and was found to furnish readings that were 10 per cent too low. When the meter was removed from the control cabinet the readings differed from those of the laboratory instrument by only 1 or 2 per cent. Bringing a piece of iron or steel up to the meter caused the reading to decrease, the amount of the decrease depending on the size, shape, and position of the metal.

The control cabinet meter has a bakelite case and the top of the control cabinet in which the meter is mounted is of steel. The steel surrounding the meter acts as a shunt for the meter's magnet, thus reducing the strength of the magnetic field around the coil of the meter and causing the reading to be low. The effect seems to be constant, since the calibration of the meter has not changed in the course of a year. Some meters in metal cases were found to be affected by iron in the same way, although the effect was much smaller than for meters in bakelite cases.

If the intensity of the radiation is measured with a suitable dosimeter, a constant error in the milliammeter reading may be of little importance, although it will give a false value of the output per milliampere. A more serious result of such an error is the danger of overloading the x-ray tube if the tube is being used at or near its full capacity. The operation of a tube at a few per cent above its rated capacity may not do any serious harm, but it is entirely possible that some meters might be affected by the presence of iron or steel so that the readings would be more than 10 per cent low, and such an overload on the tube would undoubtedly reduce its life. In view of this fact, it would seem to be advisable to check the meters without removing them from the position in which they are to be used, and the cause of any difference in the reading of the meter in the aerial system and the meter on the control cabinet should be carefully investigated. On some machines, depending on the type of rectification, the checking of the control cabinet meters would have to be done with alternating current instead of with a battery.

[1] Submitted for publication in June, 1939.

CONGENITAL CYSTIC MALFORMATION OF THE LUNGS, FORMERLY KNOWN AS "VESICULAR" OR "HYPERTROPHIC PULMONARY EMPHYSEMA," RECENTLY DESCRIBED AS "CONGENITAL CYSTIC DISEASE OF THE LUNGS," WITH REPORT OF A CASE[1]

By CHARLES TILLEY SHARPE, M.D., *New York City*

RECENT articles published on congenital cystic disease of the lungs have applied a new name to a well known and long established clinical and pathologic entity. True, x-rays have permitted the visualization of the cysts in the living subject, but they have added nothing to the excellent clinical description of the disease given by Laënnec (26) under the heading "Symptoms and Signs of Vesicular Emphysema" and also by Sir Thomas Watson (48) and others.

A study of emphysema reveals that large cysts were reported by Matthew Baillie (2), Theophile Bonet (6), Giovanni Battista Morgagni (34), Van Swiéten (46), Sir John Floyer (16), Ruisch (Friederich Ruysch), 40, and Antonio Maria Valsalva (45).

Jean Baptiste Bouillaud (7) mentioned a case in which the bladder or pouch was equal to the size of a stomach of ordinary dimensions.

More recently Charles Hilton Fagge (14), James Copland (11), Rindfleisch (37), Rokitansky (39), and Virchow (47), have reported interesting observations. For example, Fagge (14) writes:

> "There is an enormous destruction of capillaries when the affection is at all extensive. Rindfleisch speaks of the vessels as collapsing until 'only a narrow ribbon-like band is left, which may be recognized as an obliterated vessel by its greater transparency amid a dark, often pigmented, parenchyma, and by its uniting with other bands like itself to form the usual anastomotic network.' "

He goes on to say that—

> "Some relatively wide communications are opened up between the pulmonary artery on the one hand, and the pulmonary and bronchial veins on the other hand. These anastomoses appear in well injected lungs as peculiar elongated, unbranched, vascular arches of the same diameter throughout, contrasting very strikingly with a far more numerous assemblage of extremely tortuous and dilated arteries, for whose contents no such supplemental mode of escape has been provided."

Is this not an earlier description, and a satisfactory explanation, of the "fine curvilinear shadows characteristic of a multilocular cyst," described by Kirklin (24) and by Schenck (41), and visible in the case reported herewith?

As to autopsy findings, the description given by Laënnec (26) of one of his own cases is as fine a description as one is likely to find on this subject. Watson (48) gives a generalized description which agrees in many features with that of Koontz (25), as reported by Kirklin (24), and also with the description given by Schenck (41). The congenital origin of the anomaly did not escape the observation of these men, and there are arguments for and against it by some of the authors mentioned in the bibliography. For example, Sir William Osler (36) wrote:

> "On inquiry we find that these patients have been short-breathed from infancy, and they belong, I believe, to a category in which there has been a primary defect of structure in the lung tissue."

Virchow (47) was inclined to believe in the existence of a primary disease of the pulmonary tissue which developed in early life and which became more marked as time went on. He described the loss of pigment and termed it "albinism of the lungs."

The freedom of these cases of pulmonary vesicular emphysema from tuberculosis was noted by Louis (28).

The involvement of the right side of the heart in congenital cystic disease of

[1] Accepted for publication in April, 1939.

the lungs was referred to recently by Willius (49). It was described by Laënnec (26) and more extensively by Watson (48).

Many theories as to this condition were advanced and experimental work to determine the cause dates back to 1879, when Lichtheim (27) recorded his observations in the *Archives of Experimental Pathology*. More recently, Cervello (9), Hirtz (21), Friedman and Jackson (17), Harris and Chillingworth (19), and, still more recently, Hinshaw (20) report the production of subpleural emphysematous vesicles with a ball-valve apparatus, thus confirming the theory advanced by Matthew Baillie (2).

From Fantanus (15), who described "a large bladder of air which communicated with a bronchi in the lungs of a markedly cyanotic infant of three months," quoted by Meyer (33), to the case here reported, the findings are so much alike as to size, position, number, structure, contents, color within and without, time of origin, method of formation, and as to their communicating with a bronchus and at times occurring as isolated cysts, that there can be no doubt the same condition was being observed. Reports of the clinical findings by some authors, notably Laënnec (26), are so remarkably accurate that they leave no question that cases of congenital malforma-

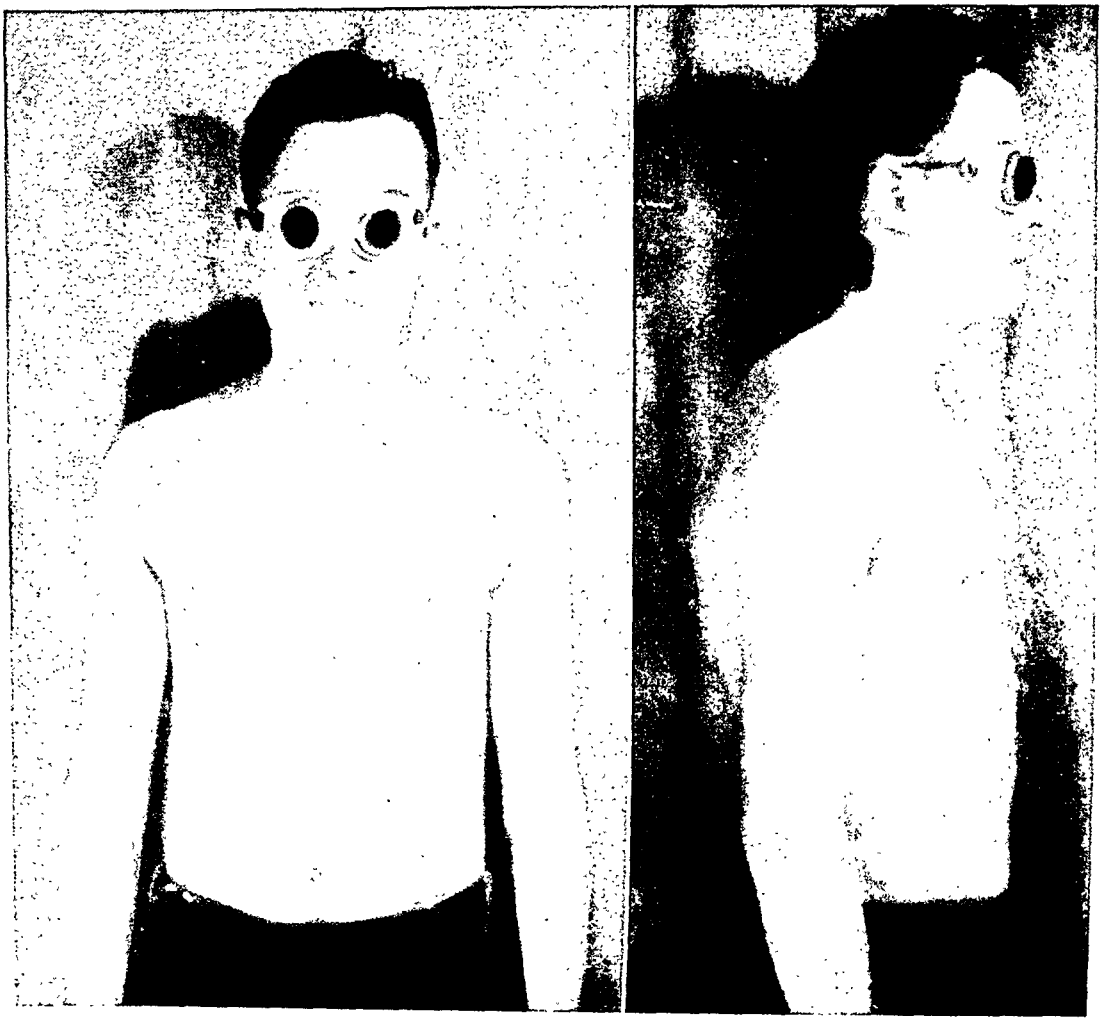


Fig. 1. The patient. Note the distention of the upper chest.
Fig. 2. Lateral view of the patient. Note the position of the spine in the upper dorsal area.

tion of the lungs were observed and well understood long before the advent of the x-ray.

Congenital cystic disease of the lungs is more descriptive than *pulmonary emphysema*, but not as satisfactory as *congenital cystic malformation of the lungs*. In selecting a new name for an old and well known condition we should not forget the dictum of Samuel Bard (4), one of the organizers of the College of Physicians and Surgeons of New York City, namely, "New names are mostly deceiving, new theories are mostly false or useless, and new remedies for a time are dangerous."

The adoption of a new name is not only often deceiving, but worse, in that it threatens the loss of the most interesting



Fig. 3. Teleoroentgenograph, postero-anterior position, during inspiration. The original film shows a marked translucency of the entire left side with curvilinear lines running in different directions, most marked longitudinally with a concavity toward the mediastinum. Quadrilateral shaped air cells are visible at the base. The entrapped air in some of these cells may be seen fluoroscopically to be behind and below the stomach. The cells change their shape with respiration. The diaphragm on the left side is not well defined because of the grouping of these air cells and the air in the fundus of the stomach. The trachea is clearly outlined to the right of the sternum. The bronchus is visible. Its branches descend almost perpendicularly to the right of the spine. The left border of the heart is hidden behind the sternum. A well defined band is seen to extend from the upper right area of the heart shadow upward toward the clavicle, terminating at the level of the second rib anteriorly on this side. This is the right border of a cyst in the right side of the chest. The axis of the ribs on the left side is more horizontal than on the opposite side.



Fig. 4. A teleoroentgenograph of the chest in the postero-anterior position, during full expiration. Note the displacement of all the contents of the right chest further to the right. The heart is much closer to the side of the cage—evidence of the pressure of the entrapped air in the left side. The curvilinear lines have changed their former position and the air cells at the base of the left chest are less quadrilateral and are extended in length. The band running from the upper right heart shadow is not visible in this film. The bifurcation of the trachea is visible and the difference in length of the right and left branches is quite evident. There seems to be some rotation of the contents in the right chest. The entrapped air is visible to the right of the spinal column. The left border of the pericardial shadow is concave. The right border of the pericardium is convex and is much closer to the side of the cage than in inspiration. During expiration the diaphragm is relatively much higher on the right than on the left, while during inspiration the two sides are about equal. The shadow of the bronchus and the aorta are confused and practically indistinguishable. This film shows a concavity of the diaphragm in the left side near the border of the cage. It is 12 cm. below the diaphragm on the right side. The compression of the lung tissue is very marked during expiration.

and valuable literature that has been compiled by our predecessors. Unless it is recognized that *congenital cystic disease of the lungs* has been known for centuries as *vesicular pulmonary emphysema*, we stand to lose as valuable a literature as we do by substituting *encephalitis* for *edema of the brain*. We might better follow the example of the dermatologists who hold fast to everything, *capillis capitis exceptis*. They add as many names as they wish in the description of dermatologic lesions, but they retain the old ones, thus preserving a complete bibliography.

Finally, if by the adoption of this new name for an old condition we sacrifice the masterful descriptions of vesicular emphysema, we may deceive our successors and limit their perspective as much as we would by failing to recognize that the great advance in our knowledge, that of visualizing the cystic changes in the lungs in life,



Fig. 5.



Fig. 6.

Fig. 5. This film was taken with the Potter-Bucky diaphragm and with the patient recumbent and in a semi-oblique position. It shows displacement of the heart shadow to the right, with a concavity in the upper area of the heart shadow due to an air cyst above it. The trachea is well defined. The bifurcation is clearly seen. On the right the bronchi are visible for a considerable length and appear to descend almost perpendicularly in the chest. The branch which appears to go to the left side is very short and seems to terminate within the entrapped air space before reaching quite to the right side of the spinal shadow. Other shadows visible in the original film suggest that the lower branches of the bronchus do not pass further to the left than the main one noted above. There is no evidence of ordinary lung markings in the left side of the chest. The axis of the ribs and their spacing are indicative of entrapped air under considerable pressure. Barium is visible in the stomach.

Fig. 6. A lateral view with the right side to the cassette. The esophagus is outlined with barium which is seen passing into the stomach. The diaphram is well defined and intact. The ballooning of the chest is evident. The upper part of the sternum bulges outward and upward, and the inner surface is markedly concave. The ribs are almost horizontal. They are widely spaced, as evidenced by their relation to the vertebral column on the left side as shown in the film. A large air cyst is visible anterior to the great vessels and the bronchi.

is due not to the work of Roentgen alone, but to a long line of scientists who devoted their attention especially to electrical research.

The bibliography which follows does not pretend to be exhaustive. It supplements in part that of Kirklin and that of Schenck.

REPORT OF A CASE

P. D., white male, examined Dec. 22, 1936; age 20 years; height 5 ft. 11¼ in.; weight 138½ pounds. Chest measurements: inspiration, 38 in.; expiration, 36 in. Abdominal measurement, 29 in. Pulse rate, 100. Blood pressure: systolic 110, diastolic 80.

Clinical Pathology.—Blood studies: hemoglobin, 94 per cent; red blood cells, 5,152,000; white blood cells, 7,600; polymorphonuclears, 75 per cent; lymphocytes, 17 per cent; eosinophiles, 3 per cent; large monocytes, 5 per cent; sedimentation rate, 5 mm. in one hour.

Urinalysis: normal.

History.—Measles at age two; otherwise negative for infectious diseases. He had some trouble with the sacro-iliac joints at age twelve or thirteen and wore a brace for one year. The pain in the back was so severe that he would fall in the street because of it.

The first indication of the developmental defect was when he started to school and it was noticed that he could not run as fast as other boys of his age. After extreme physical effort he would fall, and at times vomiting would occur. When he recovered his breath, he was all right. He has never had a serious illness.

Examination of Chest.—The outstanding symptoms and findings are barrel-shaped chest, dyspnea and cyanosis on exertion. Tactile fremitus and breath sounds are absent on the left side. Vocal fremitus is heard faintly, evidently transmitted from the right side. Râles are present at times at the right base. Excursion of diaphragm is about one inch. On fluoroscopic examination, movement of the diaphragm is seen to be accompanied by a kaleidoscopic shifting of the entrapped air present in the stomach and the air cells at the left base, some of which are below and behind the fundus of the stomach. The heart is displaced to the right side. It shifts markedly with respiration.

When examined the patient was a "runner" for a financial institution and attended evening classes several times a week. When taking part in theatrical performances he found it hard to speak his lines because of the necessity of breathing frequently. With all his handicaps, he enjoys good health and a remarkable freedom from colds and upper respiratory infection.

Every clinical detail exhibited by this case can be duplicated in the writings of the authors quoted above. The only new feature is the visualization of these cysts



Fig. 7. A view of the left side of the chest taken from the same film as shown in Figure 3 in an effort to bring out the curvilinear lines more clearly.

in the living subject, thanks to the development of the x-ray.

120 Broadway

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CAVITARY BRONCHOGENIC CARCINOMA[1]

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CHARACTERISTIC features of primary carcinoma of the bronchus are sufficiently well known to-day so that with the use of modern methods the diagnosis is in general established readily. In many instances, however, the findings so closely simulate chronic pulmonary infection that the possibility of cancer may be overlooked. This is especially true of those bronchogenic carcinomas which cavitate, since both clinically and roentgenologically they may resemble pulmonary abscess and tuberculosis. Inasmuch as carcinomas of the lung may now be amenable to surgical treatment, their exact differentiation is of practical importance. It was thus considered worthwhile to review the pertinent clinical and roentgenologic aspects of this particular type of lesion. In the American literature, special attention has been given the cavitating bronchogenic carcinomas in the reports of Fishberg and Rubin (3) and Reisner (7).

The frequency with which carcinoma of the bronchus cavitates has been pointed out by numerous pathologists from a study of autopsy material. For example, Rosedale and McKay (6) found abscess cavities in 18 of 37 autopsied cases; Jaffé (4), in 12 of 100 cases, and Fishberg and Rubin (3) in 15 of 51 cases. Koletsky (5), in a study of 100 autopsied cases, described cavitation 4 cm. or greater in diameter in 30 instances.

The pathogenesis of cavitation in primary bronchogenic carcinoma can be understood from a consideration of certain of its pathologic features. Necrosis is characteristic of nearly all tumors, resulting either from growth of the tumor disproportionate to the accompanying blood supply, or to vascular changes in or about the neoplasm. It is frequent in bronchogenic carcinoma to find the larger pulmonary vessels in the region of the hilum either compressed or invaded by tumor. Both in and around the carcinoma, smaller vessels may be found occluded by thrombi or emboli, often composed of tumor cells. These changes may, therefore, produce aseptic necrosis of tumor tissue by decreasing the blood supply. Since carcinoma of the lung arises so frequently in association with the larger bronchi, this necrotic material can be eliminated readily from the lung, leaving the pulmonary excavation.

In addition to tumor necrosis, infection also plays a significant rôle. It is well known that areas of lung distal to points of bronchial constriction are subject to various inflammatory changes. The retention of secretion facilitates invasion of the lung by pyogenic organisms from the upper respiratory tract and mouth, with the production of chronic pneumonitis and suppuration. Carcinoma of the bronchus, a frequent cause of bronchial obstruction, is prone to develop these complicating changes with suppurative necrosis of the involved tissue. As in abscess or tuberculosis, this material is expectorated with consequent cavity formation.

In occasional instances, infection of the tumor itself may be negligible, the predominant changes occurring entirely in the adjacent lung parenchyma. In these cases, the production of bronchiectasis or abscess may completely obscure the underlying carcinoma which remains undiagnosed.

The exact rôle played by necrosis of tumor tissue or infection may be difficult to determine; both mechanisms are probably involved in the majority of instances. Nevertheless, certain features may aid in

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differentiation. Tumor necrosis with cavitation is most apt to occur in those carcinomas which are infiltrative in character with a massive lobar distribution. Grossly, these cavities appear to have a thick irregular wall, the lining of which is shaggy, friable, and apparently composed of tumor tissue. The appearance suggests that the central portion of the tumor has sloughed, leaving behind a circumferential rim of carcinomatous tissue. This finding is borne out by the microscopic examination which reveals tumor at every point in the cavity wall without significant inflammatory or suppurative changes.

Cavities produced largely through infection tend to be more localized, their walls thinner and more regular, with the appearance frequently of a pyogenic membrane. The cavity contents are more purulent or fluid in character, and, depending upon the nature and extent of the infection, may be either putrid or non-putrid. Gangrene is not, in our experience, a common occurrence. Microscopic examination of the cavity wall may reveal only suppurative changes, and, it may be necessary to examine numerous sections of the wall to discover the carcinomatous infiltration.

Cavitation occurs in all types of bronchogenic carcinoma but most frequently in the squamous-cell variety. In Koletsky's (5) series, 52 per cent of squamous-cell carcinomas, 27 per cent of adenocarcinomas, and but 9 per cent of small-cell carcinomas revealed cavity formation. Similar findings are cited by Atkin (1) and Reisner (7).

Clinical Aspects.—This study is based upon a review of 127 cases of primary bronchogenic carcinoma admitted to Cleveland City Hospital from 1927 to 1937, inclusive, proved by autopsy, bronchoscopic or operative biopsy, and in which roentgenologic examination was performed. In 15 individuals (12 per cent), the roentgen demonstration of cavitation necessitated the differentiation from tuberculosis and lung abscess.

In this group the patients were all males and the ages varied from 42 to 68 years, with ten patients in the fifth and sixth decades. This finding is consistent with the age and sex distribution of all bronchogenic carcinomas at this hospital, since they predominate in males by a ratio of 9:1, while 60 per cent occur between the ages of 40 and 60 years.

A review of the histories of these 15 individuals failed to reveal any clinical picture which could be considered pathognomonic of a cavitary bronchogenic carcinoma. The important symptoms were those common also to tuberculosis and lung abscess. Cough, expectoration, loss of weight, and weakness occurred in all patients. Irregular fever above 37.5° C. was noted, at some time during the course of the illness, in 13 patients. Hemoptysis occurred in 12 cases and pain was present in 10. Five patients had clubbing of the fingers.

Although the symptomatology is usually insufficient to differentiate conclusively between these cavitating pulmonary lesions, certain aspects of the clinical picture should at least suggest the possible presence of carcinoma, particularly in individuals above the age of 40 years.

In this group of patients, the onset of the illness was insidious, with an irritative cough as the initial symptom. In eight cases, the cough was non-productive for periods as long as from three to eight months and only later was purulent expectoration noted. This latent period before the onset of expectoration is of considerable value in the differentiation from lung abscess, since patients with the latter lesion will rarely remain sputum-free for this length of time. We have not encountered a clinical picture simulating that of an acute lung abscess.

There was no type of expectoration which would be considered characteristic of a cavitary bronchogenic carcinoma. Unlike most lung abscesses, however, the daily sputum volume was small and seldom exceeded 30 c.c. of purulent material. In contrast to pulmonary abscess, foul expectoration was noted in but two patients.

Pleuritic pain is common to all cavitating lesions of the lung. In these patients with

cavitary carcinomas, however, the pain frequently appeared early, and was constant, severe, and non-pleuritic in type. Such persistent, localized pain which fails to respond to medication is unusual in abscess, and particularly in pulmonary tuberculosis, and should suggest the existence of a carcinoma.

In this group of patients, nothing of differential diagnostic value could be observed from the presence of fever, hemoptysis, weakness, fatigue, and weight loss. The physical signs elicited were variable and usually insufficient to establish the diagnosis. Obvious signs of pulmonary atelectasis and cavitation were significantly lacking. The most important finding consisted of decreased breath sounds and voice sounds in the involved area, indicating the presence of some degree of bronchial obstruction. However, similar signs accompanied by râles and impairment of the percussion note may also occur in abscess and tuberculosis. Nevertheless, in the presence of roentgenologic evidence of cavitation, diminished or absent breath sounds should indicate the possibility of an underlying carcinoma.

As in all forms of bronchogenic carcinoma, other findings may suggest the correct diagnosis. Particularly important will be the demonstration of Horner's syndrome, obstruction to mediastinal blood vessels, vocal cord paralysis, lymph node and subcutaneous metastases. However, since cavitary bronchogenic carcinomas are most frequently of the squamous-cell variety, extra-thoracic metastases will not be commonly found (5).

At the time of admission to the hospital, the clinical picture presented by this group of patients was that of a chronic, steadily progressive illness in its terminal stages. Eleven of the 15 individuals died within two months of admission. In ten of the 15 patients, the total duration of life from onset of symptoms was less than one year. One patient lived 20 months after the initial symptoms.

Roentgenologic Features.—Roentgen signs in cavitary bronchogenic carcinoma include all features observed in other types of cancer of the bronchus, with the addition of a cavity. The roentgenogram usually shows a unilateral mottled area of increased density, roughly circular or ovoid in configuration, and containing a rarefied area in or near its central portion. The extent of the density constituting the cavity wall varies from a thin line (Fig. 5) to many centimeters in thickness (Fig. 1). The ratio of the size of the cavity to the thickness of the wall depends upon the degree of necrosis and sloughing of the tumor. In our series, the signs of atelectasis were more often absent than present. Homogeneous dense areas due to pleural effusion may be an accompanying feature. The tumor cavity often contains a horizontal fluid line, usually in its base. In those instances in which the cavity cannot be recognized on the film, fluid fills the cavity completely and gives the appearance of a consolidated area similar to inflammatory or neoplastic infiltration. More frequently, however, fluid is expectorated through a communicating bronchus and the cavity remains only partially filled. A film made with greater penetration, with the Potter-Bucky diaphragm or with planigraphic technic, is often required to demonstrate the details of the cavity. The location of the tumor is variable; in our group, the cavity was situated in the right lower lobe in six instances, the right upper lobe in three, the left upper lobe in two, the left lower lobe in two, the right middle lobe in one, and along the left interlobar septum in one. If extensive, the cavity may involve more than one lobe of the lung, especially if it originates in the base of either upper lobe. Lesions near the median plane of the chest which appear on the single postero-anterior film to be located near the hilum are, in most instances, situated in the apex of a lower lobe (Cases 3 and 8). Because of the difficulty of determining the exact location of the lesion in the postero-anterior view, lateral or oblique projection is often required. Occasionally, the cavity is situated distal to the neoplasm when it does not represent the tumor (Fig. 14). The size

of the cavity ranges from the smallest diameter visible on the films to that of an entire lobe. The average size in 15 patients was 5.2 cm.; the smallest being 1.5 cm. and the largest 14 cm. in diameter. In lesions situated peripherally, with extension to surrounding structures, gross rib destruction may be observed (7). As in any type of bronchogenic carcinoma, enlarged lymph nodes may be present in the hilum as a result of regional metastasis. The cavity simulates that found in pulmonary abscess and gangrene, tuberculosis, or bronchiectasis. In the presence of a cavitary lesion of the lung, roentgenologic evidence of metastasis in bones, in brain or other soft-tissue parts may be of aid in establishing the diagnosis. Bronchograms, by demonstrating filling defects or occlusion of the lumen of the bronchus leading to the cavity, will give the clue to the diagnosis of neoplasm.

DIFFERENTIAL ROENTGENOLOGIC DIAGNOSIS

Lung Abscess.—It is almost impossible to differentiate the cavitary form of primary bronchogenic carcinoma from the ordinary type of pulmonary abscess on roentgenographic findings alone, since the appearance of cavitation with surrounding consolidation in either instance may be exactly alike. In the serial examination of lung abscess, the cavity may become gradually smaller and the consolidation resolve, while in carcinoma the lesion becomes progressively more extensive. Temporary regressions of the tumor may take place in exceptional instances (Figs. 6, 7, and 8).

Pulmonary Tuberculosis.—Carcinoma is usually a unilateral disease, whereas tuberculosis more often involves both lungs when it reaches the stage of cavitation. Mottling is usually more extensive in tuberculosis, especially in the lung which contains the cavity. Multiple cavitation is more frequent in tuberculous than in carcinomatous lesions.

Empyema.—Chronic encapsulated pockets of purulent fluid with communicating bronchial fistulæ may simulate cavitating lung tumors. Such pockets are usually located in the base of the thoracic cavity or in the periphery of the lung-fields. The shadow is usually fusiform or semi-oval in outline and is situated next to the pleural lining. Mottling is absent around the cavity. Signs of bronchial stenosis are absent.

Echinococcus Cyst.—If single, an echinococcus cyst may be confused with neoplasm (2). The cyst is smooth-walled, without infiltration in the surrounding parenchyma and without evidence of bronchial stenosis. Other foci of hydatid disease are usually present in the liver and other portions of the abdomen.

Diagnostic Procedures.—From the preceding discussion, it is apparent that in cavitary bronchogenic carcinomas neither the clinical nor the roentgenologic features are sufficiently distinctive to establish an exact diagnosis. The extent to which a correct and early diagnosis will be made must depend upon the alertness of the clinician in the utilization of the available diagnostic procedures. Early diagnosis is particularly important in this type of bronchogenic carcinoma since it is so frequently of the squamous-cell variety. Koletsky's (5) studies indicate that the squamous-cell carcinoma is slow-growing, locally invasive, and infrequently metastasizing, hence offers the best possibility for successful surgical intervention.

In our 15 patients, a correct diagnosis of carcinoma of the bronchus was made in ten instances. In one case all diagnostic procedures failed to reveal the true nature of the disease and the diagnosis was based upon the clinical course. Diagnosis was established by bronchoscopic examination in six patients and by biopsy of the lung at thoracotomy in three others. In one of the latter cases, however, the patient was considered to have a lung abscess for several months until biopsy from a deep portion of the cavity wall revealed carcinoma. Of the five incorrectly diagnosed cases, one was called sarcoma of the lung, three lung abscesses, and one pulmonary tuberculosis. In the last individual, it was known from

biopsy of a lymph node that carcinoma was present but the lung was not seriously considered as the primary source.

Bronchoscopic examination is undoubtedly the most valuable confirmatory procedure. In four of the cases erroneously diagnosed, study of the autopsy specimens indicated that bronchoscopy would probably have revealed the correct diagnosis. It has thus become an invariable rule in this hospital that all cavitary lesions of the lung which cannot be definitely established as tuberculous in origin should be examined bronchoscopically to aid in excluding the presence of an underlying carcinoma. This is especially important in males above the age of 40 years, with the clinical picture of a chronic lung abscess. Bronchoscopy will be of least assistance in diagnosing carcinomas situated peripherally or in the upper pulmonary lobes. Thus in two patients with carcinomas of the upper lobe, the trachea and bronchi appeared normal.

Occasionally, microscopic study of portions of the cavity wall removed at thoracotomy for drainage of an abscess may be of great value. In three patients the diagnosis was established in this manner. It must be remembered, however, that in those carcinomatous cavities resulting particularly from infection, the wall may show only suppurative changes and the specific section removed for examination may not reveal the presence of carcinomatous infiltration. In those instances in which abscess formation occurs entirely peripheral to the carcinoma, the cavity wall may not contain tumor tissue and the diagnosis thus remains obscure. Case 8 illustrates an example of this occurrence.

Biopsy of lymph nodes and subcutaneous nodules and the examination of pleural fluid for tumor cells may occasionally provide the clue to the correct diagnosis. Aspiration biopsy of the pulmonary lesion under fluoroscopic guidance should also be



Fig. 1.

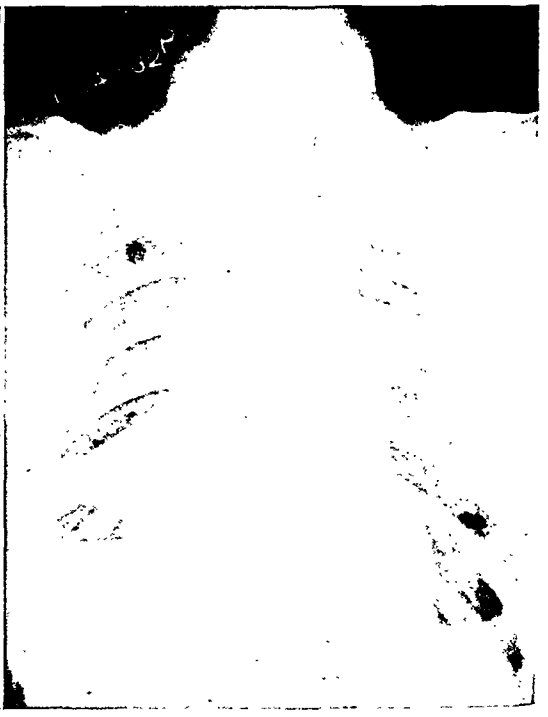


Fig. 2.

Fig. 1. Case 1. Large round mass in right base with central rarefaction representing cavity. Enlarged lymph nodes in right hilum. Original diagnosis: metastatic sarcoma. Autopsy: poorly differentiated adenocarcinoma with metastases to right hilum and right upper lobe.

Fig. 2. Case 2. Large cavity in right base with fluid level which has the appearance of a large pulmonary abscess in the lower lobe.

considered. We have employed this procedure frequently, although, in this particular group of patients, no positive results were obtained.

Repeated examination of the sputum for tubercle bacilli should always be carried out, since it will be of assistance in excluding the commonest cavernous lesion of the lung. It must be remembered, however, that carcinoma and tuberculosis may occasionally co-exist. Examination of the sputum for tumor cells was employed twice in this series as a diagnostic procedure although with negative results.

Bronchography has proven of limited value. In those instances in which evidence of bronchial constriction was present there was uncertainty whether this resulted from inflammatory change or carcinoma. The demonstration of bronchial constriction by instillation of iodized oil should, nevertheless, suggest the possibility of bronchogenic carcinoma.

ILLUSTRATIVE CASES

Case 1. J. G., a 52-year-old white male, was admitted on Aug. 3, 1928. In December, 1927, he had developed a severe cough. Two weeks later he had expectorated bloody sputum, and this recurred until the time of admission. He had lost considerable weight and complained of marked weakness. Chest pain was absent.

The patient appeared emaciated. The significant findings consisted of dullness in the right upper chest and scattered râles in the infrascapular region. The sputum was negative for tubercle bacilli.

A roentgenogram of the chest (Fig. 1) showed a circular shadow of increased density measuring 7 cm. in diameter in the lower portion of the right lung, probably in the middle lobe. In the upper portion of the shadow there was an oval rarefied area which had the appearance of a cavity. The right hilum was enlarged to 4 cm. in diameter. A small faint nodule 1 cm. in diameter was situated in the upper right lung opposite the level of the second intercostal space anteriorly. The impression was that of metastatic sarcoma.

The patient's condition was terminal at the time of admission and death occurred on Aug. 29, 1928, nine months after the onset of the symptoms. The temperature varied between 37° and 39° C.

The clinical diagnosis was that of lung tumor, probably metastatic in character. No other primary tumor could be discovered.

Autopsy (Dr. Herbert Reichle) revealed a primary carcinoma of the right middle lobe with metastases to the right upper lobe, diaphragm, right hilum lymph nodes, right adrenal, right kidney and bones. Microscopically, the tumor was a poorly differentiated adenocarcinoma. The right middle lobe consisted of a well encapsulated mass of white tissue, 10 cm. in diameter, containing a completely necrotic center.

Comment.—The smooth circular mass in the right base with the accompanying small nodule in the right upper lobe led to the erroneous diagnosis of metastatic sarcoma. Cavitation should have suggested the presence of a primary tumor, since it is rare, in our experience, to find cavitation in pulmonary metastases.

Case 2. T. P., a 49-year-old white male, was admitted on Jan. 14, 1932, complaining of a non-productive cough of several years' duration. Three months before admission, this became more marked and was accompanied by purulent expectoration. In addition, persistent pain had been present for two weeks in the right chest posteriorly. The temperature ranged between 37 and 39° C.

The percussion note was impaired over the right lower chest. Breath sounds were decreased in intensity, but numerous coarse râles were present.

Fluoroscopic and film studies of the chest (Figs. 2 and 3) showed a large cavity, 10 cm. in diameter, occupying most of the right lower lobe, but situated more posteriorly than anteriorly. The cavity was half-filled with fluid which was seen to shift under the screen while changing the patient's position. The remainder of the right lower lobe was consolidated.

The patient was considered to have a lung abscess of the right lower lobe, which was surgically drained on Jan. 22, 1932.

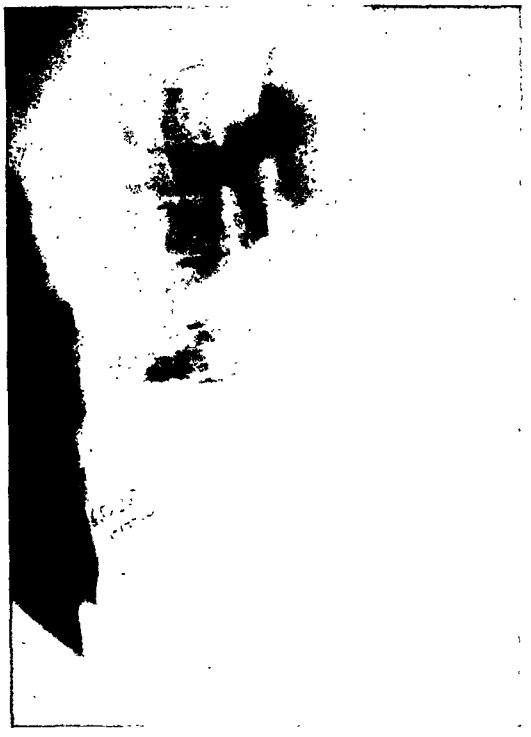


Fig. 3. Case 2. Lateral view of the chest showing a large cavity with fluid level in the posterior portion of the right lower lobe. Biopsy of the wall of the cavity showed a well differentiated squamous-cell carcinoma.

Culture from the abscess material showed gram-positive cocci and *Staphylococcus albus*. Biopsy of the cavity wall showed no evidence of carcinoma.

One month later the patient showed marked weakness, dyspnea, and loss of weight. He continued to have purulent expectoration. Partial thoracoplasty was carried out on June 2, 1932. On Oct. 10, 1932, because of his failure to improve, a biopsy was secured from the deeper portions of the abscess and this tissue revealed the presence of a well-differentiated squamous-cell carcinoma. The patient continued to lose weight, with fever ranging between 38 and 39° C. daily. Death occurred on Nov. 12, 1932, eleven months after admission to the hospital.

Autopsy (Dr. Theodore Wille) revealed a cavity 7 cm. in diameter in the right lower lobe (Fig. 4). The wall of the cavity was composed of grayish-yellow friable tissue.

Microscopically, the tumor was a well-differentiated squamous-cell carcinoma of the right main bronchus, with metastases to the regional nodes and pericardium.

Comment.—This case is representative of the type in which an original diagnosis of lung abscess was made because of the presence of a large cavity and purulent sputum. Biopsy of the cavity wall at the first operation did not reveal tumor tissue. The correct diagnosis was proven only when biopsy from the deeper portions of the abscess showed the presence of squamous-cell carcinoma. Cancer should have been suspected clinically, because of the insidious onset with a non-productive cough, in a middle-aged male, the diminished breath sounds, persistent chest pain, and failure of the patient to improve after drainage of the abscess. From the location of the tumor at autopsy, it is probable that bronchoscopic examination would have revealed the presence of carcinoma.

Case 3. J. B., a 67-year-old white male, was admitted on Sept. 7, 1933. In November, 1932, he developed cough and expectoration of small amounts of bloody purulent sputum. These symptoms were followed by weakness, persistent pain in the right chest, and weight loss of 33 pounds. Because of the clinical symptoms and the roentgen appearance of cavitation, the patient was thought to have pulmonary tuberculosis. Artificial pneumothorax had been started at another hospital.

The patient had an emphysematous chest with râles in the lower half of the right lung. The sputum was negative for tubercle bacilli. There was no elevation in temperature.

Fluoroscopic and film examination of the chest, on Sept. 12, 1933 (Fig. 5), showed a small pneumothorax on the right. There was a circular shadow of diminished density in the apex of the right lower lobe, measuring 4 cm. in diameter, due to a cavity. The infiltration forming the wall

of this cavity was thin, measuring 2 to 3 mm. in thickness.

Ten days after admission the patient died following a profuse pulmonary hemorrhage.

The clinical diagnosis was lung abscess.

The autopsy diagnosis (Dr. T. T. Frost) was squamous-cell carcinoma of the right lower lobe bronchus, with cavitation and metastases to the right bronchopulmonary and tracheobronchial lymph nodes. The lower lobe showed a 5 cm. cavity filled with blood clot and having a granular wall. Microscopically, the wall was composed of tumor tissue which invaded the surrounding bronchi, vessels, and lung parenchyma.

Case 4. H. B., a 53-year-old white male, was admitted on Nov. 28, 1934. In January, 1934, the patient developed a non-productive cough, with weakness and fatigue. In July, 1934, following the occurrence of several small hemoptyses, with a loss of 15 pounds in weight, the patient was admitted to Sunny Acres Sanatorium. During the previous month he had expectorated small amounts of purulent sputum which was not foul. Because of these symptoms and the presence of a cavity shown by roentgenographic findings, he was believed to have pulmonary tuberculosis. The sputum was persistently negative for tubercle bacilli. Since he failed to improve, he was transferred to City Hospital for further investigation.

The patient appeared emaciated. There were a few râles over the upper portion of the left lung. On the back of the neck there was a small non-tender nodule fixed to the skin. Biopsy of the nodule revealed an undifferentiated carcinoma.

Postero-anterior roentgenogram of the chest, on July 23, 1934 (Fig. 6), showed a thin-walled cavity about 4 cm. in diameter in the lateral portion of the middle third of the left lung-field. Both hili were unusually prominent. Chest films, on Sept. 8, 1934 (Fig. 7), and Nov. 7, 1934 (Fig. 8), showed a marked diminution in the size of the cavity which measured 1 × 3 cm. and was surrounded by a ring-like infiltration of greater thickness. The last examination, on Dec. 12, 1934, revealed a dense mottling, 4 cm. in vertical dimension, extending from the left hilum outward to the lateral chest wall, and a scattered nodulation from the level of the second rib to the diaphragm. There was no cavitation at this time.



Fig. 4. Case 2. Pathologic specimen showing a huge abscess in the right lower lobe and a tumor in the right eparterial bronchus.

The patient's temperature was normal. He continued to expectorate small amounts of purulent sputum. Examination revealed acid-fast bacilli on two occasions. He failed rapidly and death occurred on Jan. 29, 1935. The final clinical impression was that of pulmonary tuberculosis and carcinoma of undetermined origin. The lung was considered a possible but not proved source of tumor.

Autopsy (Dr. Mac Gallavan) revealed a small-cell carcinoma of the left lung, with metastases to the mediastinal, mesenteric, aortic, axillary and cervical lymph nodes, pancreas, spleen, and kidney. The left lung showed a firm area, 5 × 6 cm. in dimension, along the interlobar septum, containing a 2.5 cm. cavity filled with

yellow mucoid material. Tumor tissue extended along the interlobar septum to the hilum where a white mass appeared which spread into the lung in a radial manner along the bronchi and blood vessels. About 2 cm. from the carina there was slight constriction of the left main bronchus. There was no evidence of pulmonary tuberculosis.

Comment.—Cases 3 and 4 were originally diagnosed as pulmonary tuberculosis because the roentgenogram showed the presence of thin-walled cavities. Case 4 was more confusing because of the reported tubercle bacilli in the sputum and the diminution in the size of the cavity. Nevertheless, the possibility of carcinoma was suggested by the patient's progressive course, disproportionate to the roentgenologic extent of his disease, and since it was known from the biopsy of the skin nodule that he had an undifferentiated carcinoma. In Case 4 it is doubtful that bronchoscopic examination would have been of aid in the diagnosis.

Case 5. M. O., a 50-year-old white male, was admitted on June 8, 1935. Three months previously the patient had developed a non-productive cough which was attributed to a "chest cold." After several weeks he began to expectorate mucopurulent sputum which was never foul. On two occasions small hemoptyses occurred. There was persistent pain in the left upper chest. He had lost ten pounds in weight and experienced fatigue readily. Shortly before admission his sputum had become scanty.

There was slight impairment of the percussion note over the left upper chest. The breath sounds were of good quality, although slightly decreased in intensity. There were coarse râles anteriorly and posteriorly. The sputum was negative for tubercle bacilli.

A postero-anterior roentgenogram of the



Fig. 5.



Fig. 6.

Fig. 5. Case 3. A large thin-walled cavity, 4 cm. in diameter, at the level of the right hilum, was diagnosed as lung abscess. The actual location was in the apex of right lower lobe posteriorly Autopsy diagnosis: squamous-cell carcinoma.

Fig. 6. Case 4. Roentgenogram made on July 23, 1934. Arrows point to thin-walled cavity, 3 cm. in diameter, in the left upper lobe, which was diagnosed ulcerative pulmonary tuberculosis in the early course of the disease. Prominent hili.

chest on July 6, 1935 (Fig. 9), showed a dense mottling in the upper left lung-field occupying an area 7 × 10 cm. The margins of this shadow were irregular. There was a rarefied area centrally located and

Comment.—This case showed a thick-walled cavity in the upper lobe of the left lung, appearing roentgenographically as an abscess. The diagnosis of carcinoma was suspected clinically but not proven until



Fig. 7.

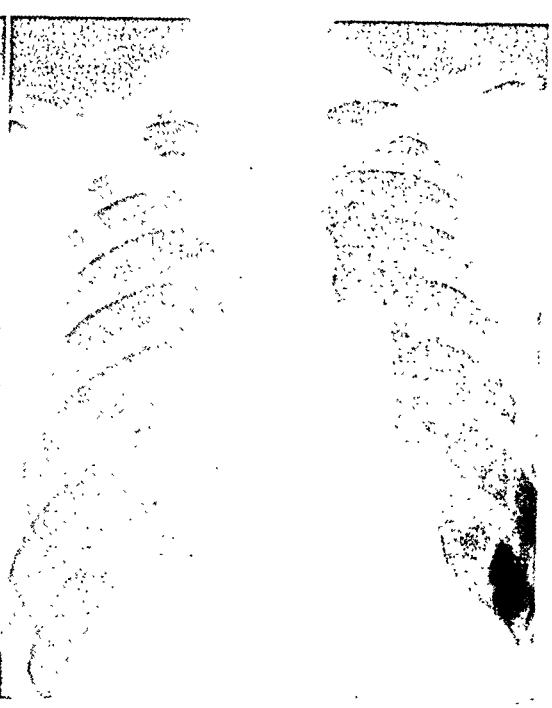


Fig. 8.

Fig. 7. Case 4. Roentgenogram made on Sept. 8, 1934, in which the cavity in left upper lobe is shown to be smaller than in Fig. 6, and surrounded by a thick ring of infiltration. The left hilum is unusually prominent.

Fig. 8. Case 4. Roentgenogram made on Nov. 7, 1934, shows the cavity to be considerably smaller than that observed on previous examination. There is a slight decrease in the thickness of the wall. Autopsy: small-cell carcinoma of the left lung with metastasis to mediastinum.

with a definite fluid level indicating the presence of a cavity.

The patient's temperature varied between 37 and 38.4° C. Because of the roentgen findings the patient was considered to have a lung abscess, possibly on the basis of a bronchogenic carcinoma. Bronchoscopy showed no evidence of intrabronchial tumor. Drainage of the abscess was performed by a two-stage operation on July 23 and July 30, 1935. Biopsy of the wall of the abscess showed the presence of a small-cell carcinoma. Following the operation, the patient failed to improve and died on Aug. 3, 1935, six months after the onset of symptoms. No autopsy was performed.

biopsy of the cavity wall revealed a small-cell carcinoma. The presence of the initial non-productive cough and chest pain were significant clinical findings.

Case 6. H. S., a 65-year-old white male, was admitted to the tuberculosis pavilion on Sept. 18, 1935, complaining of shortness of breath, cough, expectoration, loss of weight and strength for the previous three and one-half years.

The patient appeared slightly emaciated. The chest was emphysematous and the breath sounds were distant, but not abnormal. On auscultation there was prolongation of the expiratory phase. Post-tussive râles were heard at the left apex posteriorly. The temperature varied be-

tween 37 and 38° C. The sputum was negative for tubercle bacilli.

Previous roentgenographic examination of the chest on March 1, 1935, showed a streaky soft mottling in the lateral portion of the left subapical region at the level of the second intercostal space anteriorly. The appearance was that of minimal pulmonary tuberculosis. There was a moderate increase in the extent of infiltration on June 6, 1935. Because of the enlargement of the left hilum shadow, increase in the extent of the infiltration to an area 5 cm. in diameter (Sept. 24, 1935), and the appearance of large hilum lymph nodes in the lateral roentgenogram, the diagnosis of primary carcinoma of the bronchus with regional lymph node metastases was considered. The right anterior oblique view on Oct. 8, 1935 (Fig. 10), and the postero-anterior view on Nov. 9, 1935 (Fig. 11), showed a definite cavity with a fluid level in the center of the consolidation. On Jan. 23, 1936, ten days before death, the consolidation occupied the upper third of the left lung-field.

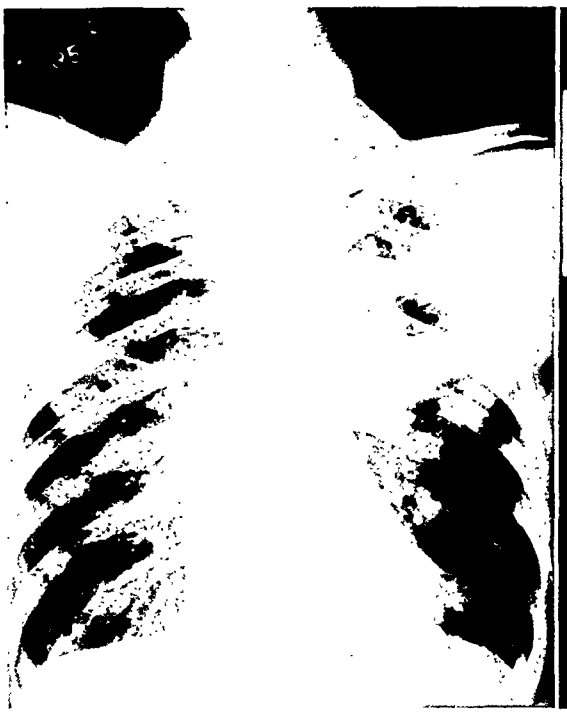


Fig. 9.

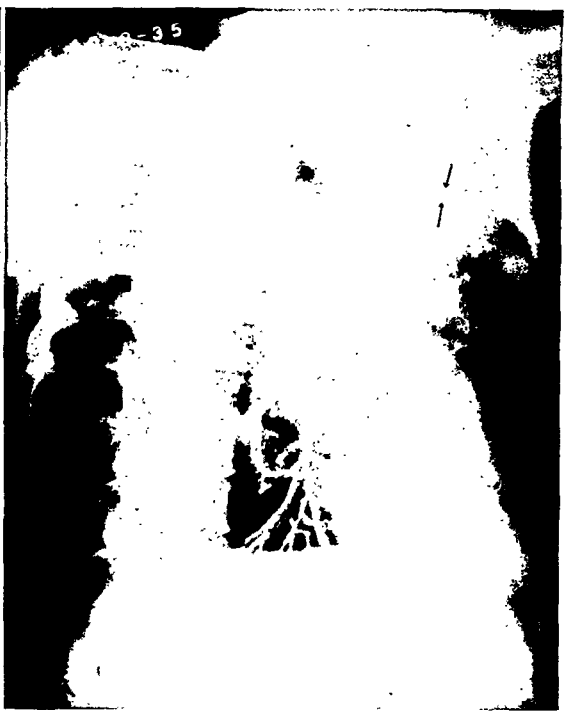


Fig. 10.

Fig. 9. Case 5. A cavity, 5 cm. in diameter, containing fluid and surrounded by dense infiltration in the right upper lobe. Clinical and roentgen diagnoses: lung abscess. Biopsy of the wall of the abscess showed small-cell carcinoma.

Fig. 10. Case 6. Right anterior oblique view of the chest (Oct. 8, 1935) showing a small cavity, 1.5 × 3 cm., in the left subapical region surrounded by dense infiltration. At this time the cavity was not visualized in the postero-anterior view.

Bronchoscopic examination on Oct. 8, 1935, was negative. Iodized oil failed to enter the left upper lobe. Aspiration biopsy was attempted on two occasions without success. The patient developed bradycardia, suggesting metastasis to the brain. He became extremely nervous and confused mentally. Death occurred on Feb. 2, 1936. The final clinical diagnosis was carcinoma of the bronchus.

Autopsy (Dr. A. Chernoff) revealed a poorly differentiated adenocarcinoma of the left eparterial bronchus with cavitation and metastases to the left bronchopulmonary lymph nodes, right adrenal gland, and brain. In the left upper lobe a cavity

6.5 cm. in diameter was found (Fig. 12). There was a soft mass growing down into the bronchus from the edge of the cavity about 4 cm. from the left main bronchus.

Comment.—Case 6 represents a thick-walled cavernous lesion of the lung. The patient was admitted with a diagnosis of pulmonary tuberculosis because of the original roentgenographic appearance. However, the enlargement of the left hilum, the persistent negative sputum, and the progressive course established the diagnosis of bronchogenic carcinoma. Bronchoscopy, bronchography, and aspiration biopsy all failed to indicate the true nature of the lesion.



Fig. 11. Case 6. (Nov. 9, 1935.) A large circular area of increased density, measuring 4.5 cm. in diameter, in the left subapical region showing at this time a small cavity in its central portion. The arrow points to fluid level. (See postmortem specimen.)



Fig. 12. Case 6. The postmortem specimen showing large round tumor in the left apex. The central portion of the neoplasm contains a cavity the walls of which are lined with necrotic material. Microscopic examination revealed a poorly differentiated adenocarcinoma of the eparterial bronchus.

Case 7. A. C., a 54-year-old white male, was admitted on July 17, 1936, complaining of severe cough for the previous five and one-half months. There was no purulent expectoration or hemoptysis. For five weeks previous to admission, the patient had had pleuritic pain on the right side. The patient showed evidence of weight loss and had a persistent irritative dry cough. The chest was dull to percussion over the lower half of the right lung posteriorly. Breath sounds were absent and scattered râles were heard in the right midlung anteriorly and posteriorly. The temperature was 39° C. The sputum was negative for tubercle bacilli. On aspiration, 775 c.c. of thin purulent fluid with a specific gravity of 1.022 were obtained. No organisms were seen on the smear. Histologic examination of the pleural fluid revealed no tumor cells.

Thoracotomy on July 28, 1936, was followed by considerable improvement. The patient was lost to observation until Aug. 3, 1937, when it was found that copious foul expectoration had developed,

with 20 pounds loss of weight and constant pain in the right chest. Thoracentesis produced 60 c.c. of pus. Surgical drainage of a large bronchiectatic cavity in the right lower lobe was done on Aug. 7, 1937.

pleural effusion. Bronchograms made on Jan. 21, 1937 (Fig. 13), showed cylindrical dilatation of the lower lobe bronchi as a result of bronchiectasis. No significance was attributed to the constriction at the origin

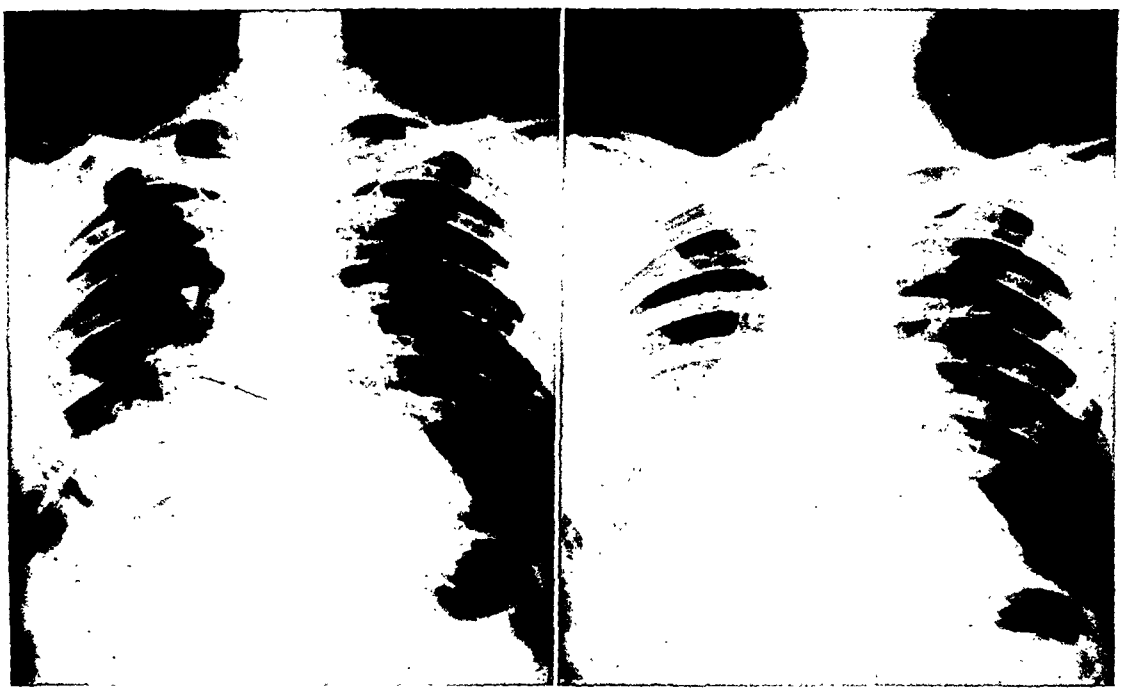


Fig. 13. Fig. 14.

Fig. 13. Case 7. (Jan. 21, 1936.) Instillation of iodized oil in the right lower bronchial tree. Arrows show constriction of the right descending bronchus at the site of the carcinoma. Lower bronchi show cylindrical bronchiectasis. Inflammatory reaction in the right lower lobe in the area of the surgically drained abscess.

Fig. 14. Case 7. (Aug. 4, 1937.) A large cavity with fluid level in the right lower lobe, posteriorly, is shown. The cavity represents an abscess distal to the neoplasm which is situated at the proximal portion of the right main descending bronchus. Resection of the right rib in posterior axillary line.

Biopsy of the pleura and underlying lung showed chronic inflammation. The patient's condition became progressively worse and he expired on Oct. 12, 1937.

Roentgen examination of the chest on July 16, 1936, revealed a dense homogeneous cloudiness throughout the lower half of the right lung-field with an ill defined upper border, indicating the presence of fluid and consolidation. After partial resection of the ninth right rib, a small pneumothorax was present with a moderate collapse of the right lower lobe. On Nov. 17, 1936, the cloudiness in the right base extended higher laterally than medially and represented a re-accumulation of the

of the right descending bronchus. Postero-anterior and lateral views on Aug. 4, 1937 (Figs. 14 and 15), showed a large cavity in the right base with an extensive amount of infiltration surrounding it.

Autopsy (Dr. Simon Koletsky) showed the fistula in the right chest to lead into a large irregular cavity occupying the postero-lateral portion of the lower lobe, a portion of the middle lobe, and a small portion of the upper lobe adjacent to the hilum. The fistula extended into the pericardium. A tumor mass involved the right lower lobe bronchus about 1 cm. from its origin and extended into the pulmonary tissue to the eparterial bronchi. Micro-

scopic examination showed a poorly differentiated squamous-cell carcinoma of the lower lobe bronchus with extension into the right lung, the superior and inferior bifurcation lymph nodes, and pericardium.

bronchus on the bronchogram was masked by the cylindrical bronchiectasis. Bronchoscopy should have been performed and would have revealed the nature of the lesion.



Fig. 15. Fig. 16.

Fig. 15. Case 7. (Aug. 4, 1937.) Lateral view showing a large cavity with fluid level in the posterior portion of the right lower lobe. Abscess distal to the carcinoma.
Fig. 16. Case 8. A cavity, 4 cm. in diameter, with fluid level, situated in the apex of the right lower lobe. (See gross specimen in Fig. 17.)

The cavity wall revealed tumor infiltration only in its medial portion. The lateral wall adjacent to the site of operation showed no tumor tissue.

Comment.—Case 7 illustrates the type of case in which the abscess cavity lies entirely peripheral to the carcinoma and hence obscures it. In these instances it can be understood why biopsy of the cavity wall may not be of diagnostic value. Carcinoma was not suspected in this individual although the insidious onset with non-productive cough, decreased breath sounds, and the progressive course suggested its presence. The significance of the constriction of the right lower lobe

Case 8. J. S., a 68-year-old white male, was admitted on July 25, 1936. In February, 1936, the patient had developed a cough with slight blood-streaked sputum. Since that time he had lost 40 pounds in weight and complained of weakness.

Examination showed the patient to be emaciated. There was slight retraction of the right supraclavicular fossa. The breath sounds in both lungs were obscured by sonorous râles.

A postero-anterior view of the chest on July 27, 1936, showed a cavity 2 cm. in diameter in the right hilum region and dense mottling in the medial portion of the lower third of the right lung. On Sept.



Fig. 17. Case 8. Autopsy specimen showing large cavity in the apex of the right lower lobe. Microscopic examination showed a well-differentiated squamous-cell carcinoma.

15, 1936 (Fig. 16), re-examination of the chest including a lateral view showed the cavity to be located in the upper posterior portion of the right lower lobe and to have increased in size to 4 cm. in diameter. A definite fluid level was present at this time.

In the differential diagnosis, carcinoma, bronchiectasis, and tuberculosis were considered. Bronchoscopic biopsy obtained from the right lower lobe bronchus on Sept. 11, 1936, revealed a well-differentiated squamous-cell carcinoma.

The patient's condition became progressively worse. The temperature ranged between 37 and 38° C. Death occurred on Sept. 22, 1936, after three days of profuse hemoptyses.

At autopsy (Dr. Simon Koletsky) a cavity 6.5 cm. in diameter was found in the upper posterior portion of the right lower lobe (Fig. 17). The cavity wall consisted of friable necrotic tissue. The cavity communicated with the right main bronchus. The walls of the main bronchus, eparterial and hyparterial bronchi were the site of the tumor formation. Tumor tissue extended along the bronchi into the middle and lower lobes. There was direct extension of tumor into the adjacent hilum lymph nodes. Microscopically, the wall of the cavity showed a well-differentiated squamous-cell carcinoma.



Fig. 18. Case 9. Postero-anterior view of the chest shows a large area of cavitation in the upper half of the right lung. Note the shaggy appearance of the upper portion of the wall. Fluid level in the base of the cavity. This is the largest cavitating carcinoma in this series.

Case 9. J. P., a 64-year-old white male, was admitted on Jan. 14, 1937. The patient had noticed a dry cough for 15 months. Nine weeks before admission it had become severe with production of mucopurulent expectoration. Fatigue, weakness, and loss of 15 pounds in weight had occurred.

There was dullness to percussion over the right upper chest with decreased breath sounds and a marked post-tussive suction. Coarse râles were heard over the entire right lung. The sputum was negative for tubercle bacilli.

Postero-anterior and lateral roentgenograms of the chest made on Jan. 12, 1937, revealed a hugh cavity 14 cm. in diameter in the posterior portion of the right upper lobe (Fig. 18). The cavity contained a fluid level. In the lateral view the cavity was seen to lie somewhat obliquely and to be oval in outline, with its lower pole extending anteriorly and the upper posterior margin at the chest wall. The infiltration forming the wall of the cavity was about 1

cm. in average thickness. The inner border of the upper lateral wall of the cavity was shaggy in outline.

The patient continued to have profuse foul sputum. The lesion was considered to be a lung abscess although the possibility of a bronchogenic carcinoma was also mentioned. On Jan. 18 and 22, 1937, a two-stage drainage of the abscess was performed. Biopsy of the wall showed a moderately well-differentiated squamous-cell carcinoma. After operation the patient failed to improve and died on Feb. 8, 1937. Autopsy was not performed.

SUMMARY

Cavitation occurs frequently in bronchogenic carcinoma resulting from necrosis of tumor tissue, infection, or both. In a series of 127 proven cases, 12 per cent had roentgenologic evidence of cavity formation.

Cavitary bronchogenic carcinomas are often confused with lung abscess and tuberculosis since neither the clinical nor roentgenologic pictures are sufficiently characteristic to establish a conclusive diagnosis.

In the presence of roentgenologic evidence of cavitation, certain findings suggestive of carcinoma may be elicited. Of importance will be the history of an insidious onset, non-productive cough, persistent pain, diminished breath sounds, and bronchographic evidence of bronchial constriction.

Accurate and early diagnosis of cavitary bronchogenic carcinoma depends upon the utilization of the available diagnostic procedures. Of those discussed, bronchoscopic examination is of greatest value, particularly in the differential diagnosis of cavitary pulmonary lesions in patients above the age of 40 years.

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PRIMARY SARCOMA OF THE STOMACH

REPORT OF THREE CASES[1]

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THE stomach is a rare seat of the sarcomas, but they probably are more frequently present than recognized, because of the lack of characteristic clinical symptoms and roentgen diagnostic signs, and the fact that not every gastric tumor, surgically removed, undergoes microscopic study. Even the pathologist may misdiagnose a gastric sarcoma on account of its close resemblance to anaplastic (embryonal) forms of carcinoma (3) and other conditions more frequently found in gastric lesions. Freeman (7) reported a case of gastric sarcoma in which prominent pathologists gave a diagnosis of carcinoma, lymphosarcoma, inflammation, and chronic granuloma.

The first gastric sarcoma was reported by Bruck, in 1847, and the first operation was performed by Virchow, in 1887.

The frequency of gastric sarcoma is about 1 per cent of the malignant gastric conditions, as estimated by Ewing (6), Phillips and Kilgore (14). The ratio was 1 : 121 at the Mayo Clinic (1) during the period from 1908 to 1928. In 4,509 malignant gastric tumors operated upon in seven large clinics in various parts of the world, the sarcoma–carcinoma ratio was found to be 1 : 93. Only one gastric sarcoma has been reported in the Berlin Pathologic Institute (11) among 840 specimens of sarcomas.

The age sector of gastric sarcoma is wider than that of carcinoma; there are cases on record in every decade of life up to the ninth. The extremes are Finlayson's three-and-one-half-year-old and Giacomma's ninety-one-year-old patient. The average age incidence is 43 years, at the Mayo Clinic (1); other reliable statistics state similar age incidence, the most frequent decade being the fourth (9). These data verify the appearance of gastric sarcoma earlier in life than that of carcinoma.

Most of the statistics report an even distribution of cases in both sexes. At the Mayo Clinic, males are affected three times as frequently as females.

Histologically, the gastric sarcomas are classified as fibro-, myo-, lympho-, and myxosarcoma. Fibrosarcoma originates in the submucosa; myosarcoma in the muscularis; lymphosarcoma in the lymph nodes of the submucosa; subserous origin of these sarcomas is less frequent (13).

Clinically, they may be exo-, endogastric, and intramural tumors; well defined or diffuse growths. The exo- and endogastric types are sometimes pedunculated. Fibro- and myosarcoma arise mostly from the curvatures; pyloric involvement is rare. Brodowsky reported a tumor the size of a child's head; Cantwell, one weighing 12 pounds. Also exceedingly small tumors are described. Endogastric growths sometimes undergo ulceration and may cause severe, even fatal, hemorrhages. These tumors usually grow slowly, metastasize late, and form a well-defined mass. Lymphosarcoma involves the pylorus or the curvatures and may produce a diffuse growth but may protrude into the gastric or abdominal cavity as an endo- or exogastric tumor; stenosis has been reported, but it is not common (15). Lymphosarcoma metastasizes early and spreads to the neighboring organs by local extension (8).

The clinical symptoms reported in the literature usually are the same or just slightly deviated from the symptomatology of the carcinoma, but sometimes the addition of clinical symptoms, roentgen signs,

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and laboratory findings may be suggestive of a gastric sarcoma.

The general health and condition of the individual is less affected in gastric sarcoma than in carcinoma (2). (It is interesting that we also noticed this to be true in our series of 36 osteogenic sarcomas.)

In some cases the classical symptoms of a gastric or duodenal ulcer are dominated with pain-food-ease rhythm (9).

The general gastric symptoms of malignancy—indigestion, hemorrhage, vomiting—occur later (4) than in carcinoma because the mucosa is only secondarily involved.

Copious hemorrhages with recovery and remissions are a suggestive sign of an ulcerative sarcoma.

Palpable tumors, sometimes huge in size, were present in about 50 per cent of the cases reported by Eusterman and Balfour (5); in some cases they are the leading signs; a large tumor without marked symptoms of malignancy is highly suggestive of sarcoma. The mobility or immobility of the masses has no significance, since freely movable, immobile, and tumors moving with respiration are noted in the case reports.

The blood picture may remain closer to the normal than in carcinoma, although definite secondary anemias have been reported.

The roentgen diagnosis of gastric sarcoma is difficult and is discussed as much as its symptomatology. Golden (8) believes that a well-defined filling defect with a crater shadow in it is suggestive of a sarcoma. He has seen three proven cases of gastric sarcoma with this roentgen finding. Grier (10) states that sarcoma is characterized by a much larger tumor than is carcinoma.

Kandruka and Sierro (5) believe that a pedunculated endo- or exogastric tumor is strongly suggestive of a sarcoma. The pedunculated endogastric tumor is characterized by a sharply defined rounded filling defect with relative mobility and a constant notch at the base of the tumor corresponding to the implantation of the pedicle. The exogastric type produces a narrow pressure defect on the stomach wall at the base of the pedicle with more or less displacement of the organ. Peristalsis may be diminished but not stopped at the attachment, and mucosal folds are not disturbed markedly. These investigators believe that in most cases a lymphosarcoma cannot be differentiated from carcinoma roentgenologically.

Spitzenberger (15) reports a case of lymphosarcoma which originated from the fundus, extended through the diaphragm, and affected the function of the heart. He believes that sarcomas of the upper end of the stomach originate from the greater curvature side of the fundus; carcinomas from the lesser curvature side of the cardia.

Kessler (15) made considerable study of the roentgen appearance of gastric sarcomas and she believes that a flat tumor with smooth borders, a tumor with deeply indented relief, or any single tumor on the greater curvature lacking the characteristic saucer-shape of carcinoma of this location is suggestive of a sarcoma.

The well defined exo- and endogastric sarcomas are best treated surgically—post-operative series of roentgen therapy is quite helpful. The infiltrating types and inoperable cases should be treated by radiation therapy. Extreme caution is required in the dosage of roentgen therapy of gastric sarcoma because of the high radiosensitivity of the tumor. The rapid absorption of the tumor products may cause severe toxicosis, even death. A fatal gastric hemorrhage may occur by the sudden perforation of a larger gastric artery, which was a constant discouraging danger in the pioneer experimentation of roentgen therapy of inoperable gastric carcinoma (12).

To avoid these fatalities, one should divide the area to be treated into small fields and use small daily and total doses. Similar caution should be applied in case of post-operative roentgen treatment of gastric sarcomas.

CASE REPORTS

Case 1. White female, 61 years old, first noticed slight pain in the epigastric region about nine months ago. The pain and discomfort came on about two hours after eating and continued until the next feeding; was easily relieved by food. Occasionally there was gas on the stomach. She had no nausea, vomiting, nor loss of appetite. The patient had lost only a few pounds in weight since first symptoms. She did not notice anything peculiar about her stools, but had had moderate constipation for years. One sister had died of carcinoma; otherwise the family history was not interesting.

Physical Examination.—A poorly nourished, white, elderly female, anemic but not acutely ill. Head and neck essentially negative. Respiration regular, expansion equal; no râles. Heart size and sounds, normal. Blood pressure, 150/100; vessel walls sclerotic. There was a hard, tender, irregular, movable mass in the right epigastrium about seven centimeters in diameter. Extremities and genitalia were normal. Behavior was rational and cooperative. Temperature at admission, 98.2° F.

Laboratory Findings.—Gastric content: mucus, heavy; bile, negative; blood in faint trace; total acid 22, free HCl, 12; red blood cells, 2,980,000; hemoglobin, 58 per cent (Sahli); white blood cells, 8,100; neutrophiles, 84 per cent; blood, Type II; Kline test, negative.

Roentgen Examination.—A stomach of normal size and position was shown. There was a large irregular filling defect at the distal third of the greater curvature, which did not involve the pylorus. Peristalsis was active and stopped at the filling defect. The duodenum was normal. At five hours there was some staining in the stomach and duodenum; the lower ileum was filled. At 24 hours, the transverse and descending colon was filled. Roentgenography of the chest revealed no evidence of metastasis.

A provisional diagnosis of gastric carcinoma was made. The patient received a total amount of 1,600 c.c. citrated blood within a week, which raised the red blood count from 2,980,000 to 4,150,000, and the hemoglobin from 58 to 78 per cent.

On operation, a large growth was found situated in the prepyloric region on the greater curvature. Glands were found in the gastrohepatic and gastrocolic region. The liver, aortic and lumbar glands were negative. There was no attachment to the pancreas or colon.

Microscopic Diagnosis.—Small round-cell sarcoma, probably lymphosarcoma.

The patient made an uneventful recovery, was discharged in good condition on the twenty-fourth post-operative day, and was receiving a series of post-operative roentgen-ray therapy.

Case 2. About seven months ago, this patient, a 65-year-old white male, began to have gas on the stomach and epigastric pain which was relieved by belching. This usually occurred after meals, but he was bothered at other times also. Pain was relieved by belladonna several times at the onset of symptoms. There was no noticeable change in his condition until about one month ago, when all his symptoms became more severe and the patient consulted his physician, who prescribed a milk diet. The patient began to vomit immediately after each meal and had the gas and pain as before. There was no blood in the vomitus, which the patient described as tasting neither bitter nor sour, just "awful." His doctor advised him to have an x-ray examination to make sure of the diagnosis. The patient had also complained of constipation for the past seven months, although there was nothing abnormal about the form or color of stool. There was no jaundice. Average weight, 126 pounds; maximum weight, 135 pounds; present weight, 105 pounds.

Physical Examination.—A well developed, poorly nourished, white male, 65 years of age, slightly cachectic. Not acutely ill. No gross abnormalities. Head and neck essentially negative. Chest:



Fig. 1. Case 1. Filling defect with deep indentation on greater curvature.
Fig. 2. Case 3. Constant narrowing of the prepyloric part.

expansion good and equal, breath sounds normal. Heart: blood pressure 126/70; normal in size, shape and position, and rhythm; no abnormal valve sounds. Abdomen: entire abdomen felt soft and doughy except for an indefinitely outlined mass or a spasticity in the epigastrium filling the costal angle; no tenderness. Rectum and genitalia showed nothing remarkable. Extremities: reflexes were sluggish, otherwise, normal.

Roentgen Examination.—Stomach: J-shaped, lower pole was somewhat lower than normal. No filling defects. Peristalsis and duodenal cap were normal. There was a pressure defect on the greater curvature side of the pars media. Slight gastric retention in five hours. Twenty-four- and 48-hour films showed normal advancement of the barium. Diagnosis: Pressure defect caused by an exogastric tumor. The patient was unable to retain barium enema

Laboratory Findings.—Red blood cells, 5,280,000; hemoglobin, 90 per cent; white blood cells, 11,450; neutrophiles, 84. Gastric content: total acid, 35; free HCl, 15; blood, 0, bile, 0, mucus, moderate. Feces: blood in traces; fat, bile, ova, parasites, negative. Urine, negative. Temperature at admission, 97° F.

Exploratory laparotomy was advised, but the patient refused it, left the hospital, and re-entered four weeks later. The mass now filled the entire upper mid-abdomen, was irregular in outline, semi-fluctuant, tender, and slightly movable. Movement caused pain. Since leaving the hospital, the patient had vomited several times daily, had had black stools, poor appetite, and continued loss of weight and strength. He was now emaciated and willing to submit to operation. Exploratory laparotomy was done on Sept. 22, 1933. On opening the peritoneal cavity, a large hard mass was found in the stomach adherent to the large intestine and pancreas. The patient's condition did not permit further exploration. A small nodule was excised from the greater omentum for biopsy, and the wound was closed in the normal manner. The patient gradually failed and died two days later. Autopsy was not performed.

Microscopic diagnosis of the tissue removed was that of lymphosarcoma.



Fig. 3. Case 3. The stomach is normal.

Case 3. This patient, a three-and-one-half-year-old white male, weighed 10.5 pounds at birth. He had lost weight and had not done well on breast milk; had had vomiting spells, at which time he would vomit two days at a time. He had not done well on bottle feeding and had gained very little weight until he began eating the regular diet of a child. The patient had had chicken pox, measles, and whooping cough without complications. He took cold easily. The family history was negative—no accidents nor operation.

The patient had had pneumonia in February, 1932, was ill about one week, made an uneventful recovery but complained of pain in the region of the umbilicus. This pain disappeared after pneumonia but cramping attacks returned two months later, in April. The patient had frequent colds and would complain of pain in the region of the umbilicus associated with each cold. He was always constipated. In November, he had one vomiting spell which lasted four days. There was marked increase in the severity of constipation, with only one bowel action in four days sometimes. He complained of some pain in the kidney regions, and the urine was dark yellow in color. In December, patient began to complain of severe attacks of pain in the abdomen, and he was admitted to the hospital three weeks later.

In the hospital, examination of the abdomen showed some tenderness along the colon and superior to the umbilicus. No masses were palpable. On the second day of hospitalization, severe paroxysmal cramping pain developed in an area just above the umbilicus. This was not relieved by enema, paregoric, or atropine. Patient vomited after onset of pain. Severe vomiting was seen two days later with paroxysmal pain.

Roentgen Examination.—The gastro-intestinal tract revealed a constant narrowing, immediately prepyloric, with extreme gastric retention in five hours and some retention shown on the 24- and 48-hour films. There was also evidence of considerable partial obstruction high in the small intestine indicated by a markedly dilated small bowel loop.

The patient was operated upon the following day for intestinal obstruction. Upon opening the abdomen, a purple-colored distended intestinal mass presented itself in the right upper quadrant and epigastrium. This was gradually manipulated out from the abdomen and found to be a volvulus of jejunum and ileum with about twelve inches of the intestine twisted. When the volvulus was untwisted and the adhesions broken up, the mesentery was found to contain many discrete hard tumors from 0.5 to 1 in. in diameter. There were some clusters of these tumors and, apparently, their weight had caused the volvulus. One of these tumors was removed for biopsy. The patient made an uneventful recovery.

Microscopic Examination.—The node removed showed a lymphoblastic process, apparently superimposed on some low-grade chronic type of inflammation. Microscopic diagnosis: lymphoblastoma, probably lymphosarcoma and chronic lymphadenitis.

Fourteen days after operation, a course of roentgen therapy was administered to the upper abdomen. Three months later, the patient returned for a check-up examination, was practically symptomless, but roentgen examination of the gastro-intestinal tract still revealed a constant rigid narrowing of the prepyloric part; five-hour film showed slight gastric retention, otherwise normal advancement of the barium; 24-hour film showed barium to be distributed in the colon. The previous roentgen treatment was repeated. Since the second course of roentgen therapy, the patient has remained symptomless, developed normally, and the last roentgen examination, done six and one-half years after the first irradiation, revealed a stomach normal in shape, size, and position, with mucosal folds, peristalsis, and duodenal cap regular. At five hours, there was no gastric retention, the lower ileum was filled with the barium; at 24 hours the barium was distributed in the colon.

Summary of Case Reports.—Three cases were reported from our series of gastro-intestinal sarcomas, in which the diagnosis and localization of the primary lesion were verified by microscopic and x-ray examinations and also by surgical exploration. The cases in which the diagnosis or origin of primary lesion was doubtful are not mentioned in this report.

One of our patients is probably the youngest individual (three and one-half years old) having gastric sarcoma to have been reported in the literature; the other two are in the sixth decade.

Case 1 had typical peptic ulcer symptoms with pain-food-ease rhythm of nine months' duration, without nausea or vomiting. She had a large endogastric tumor with deeply indented relief, indicating destruction of the mucous membranes, resulting in occult bleeding, anacidity, and anemia.

Case 2 had fairly typical ulcer symptoms of nine months' duration with nausea and vomiting in the last month. Since his tumor was exogastric, the gastric mucosa was not involved, consequently the blood picture was normal, gastric acidity only slightly lowered, occult bleeding only in traces.

Case 3, we believe, is one of the youngest patients having gastric sarcoma reported in the literature. Although a diagnosis of sarcoma of the stomach was made by roentgen examination alone, we admit that the age of the patient was of considerable importance in differentiating it from carcinoma. Summing up the history of unusual frequency of vomiting, indigestion starting soon after birth, constancy of gastric symptoms, disseminated metastasis at three and one-half years of age, we believe this is a case of congenital lymphosarcoma of infiltrating type.

Conclusion.—Pre-operative diagnosis of a gastric sarcoma is extremely difficult, since there are no characteristic symptoms and signs to differentiate it from carcinoma.

The age incidence of gastric sarcoma is earlier than that of carcinoma, a gastric tumor in the first three decades of life being highly suggestive of a sarcoma. The duration of symptoms is usually shorter than that of carcinoma.

A large tumor with diminutive symptoms of malignancy is strongly suggestive of a sarcoma. Gastric sarcoma may simulate a peptic ulcer symptomatically.

Hemorrhage with recovery and recurrence without marked anemia is a suggestive sign. Loss of weight is noted less frequently in sarcoma than in carcinoma.

Roentgenologically, a flat tumor with smooth borders, pedunculated growth, isolated filling defect on the greater curvature with deep indention or with a crater shadow in it, and tumors on the greater curvature side of the fundus, are suggestive of sarcoma. Due to the radiosensitivity of sarcoma, an inoperable tumor may be rendered operable after a course of roentgen therapy and in some instances the tumors are curable by radiation alone.

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RESPONSES OF *DROSOPHILA* PUPAE TO X-RAYS[1,2]

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EGGS of *Drosophila melanogaster* have been found by a number of investigators, particularly Packard (1), to possess properties which make them favorable as test objects for radiobiologic investigations. They are small, comparatively radiosensitive, usually easily obtainable, and manifest a clear-cut response to radiation (hatching or failure to hatch). However, as pointed out by this laboratory (Henshaw and Henshaw, 2), and by Packard (3), a marked variation in radiosensitivity occurs with age, in these forms, and it is difficult, if not impossible, to control the age factor adequately in experiments involving collection and treatment intervals of more than a few minutes.

Mavor (4), in 1923, pointed out that *Drosophila* pupæ have a period of several hours during which their radiosensitivity is nearly constant. Since we had need for new test material for quantitative radiobiologic work, experiments were carried out to determine the suitability of these organisms. At the outset we may say that they were found suitable and that a paper from this laboratory has been published already, based on work involving their use (Müller, 5). Although several times larger than eggs, the pupæ are, nevertheless, small and convenient to handle, are easily obtainable, resistant to drying, uniformly sensitive to radiation for a number of hours, and, like the eggs, give a clear-cut response to radiation (*i.e.*, they either emerge or fail to emerge as imagos). It is the purpose of this report to present the ground work experiments which justified the use of this organism as test material as well as to present some of the interesting radiobiologic relationships which have been observed.

The following will be dealt with particularly: (1) a description of pupal (and pre-pupal) development as related to the problem at hand, including methods of collecting, restricting age, and identifying stages of development; (2) changes in radiosensitivity with age; (3) the relation between radiosensitivity and developmental activity; (4) reproducibility of the quantitative response of pupæ to x-rays, and (5) the influence of lethal doses of x-rays on respiratory rate.

1. *Pupal Development.*—Starting with the freshly fertilized egg of *Drosophila*, the adult stage is reached in about ten days when maintained at 26° C. The egg develops along lines typical of insects, in general, and hatches into a larva in from 18 to 24 hours. The larva is a moving form which feeds actively and grows continuously. After about five days of larval activity, the organism ceases feeding and crawls up the sides of the culture bottle in preparation for becoming a pupa. After crawling about for several hours, movement begins to decrease and finally stops altogether. As activity ceases, the oral hooks are withdrawn, the anterior spiracles are everted, and a thin chitinous case, or *puparium*, is secreted about the organism.

Since the present investigation was first undertaken, a detailed embryologic account of pupal (and pre-pupal) development of *Drosophila melanogaster* has been published by Robertson (6). The reader is referred to this for additional information. Robertson has pointed out, with justification, that in order for *Drosophila* development to be consistent with that established for other insects, true pupation does not occur until about 11½ hours after cessation of movement. However, since this technical embryologic consideration does not concern us here and since we were interested in the complete quies-

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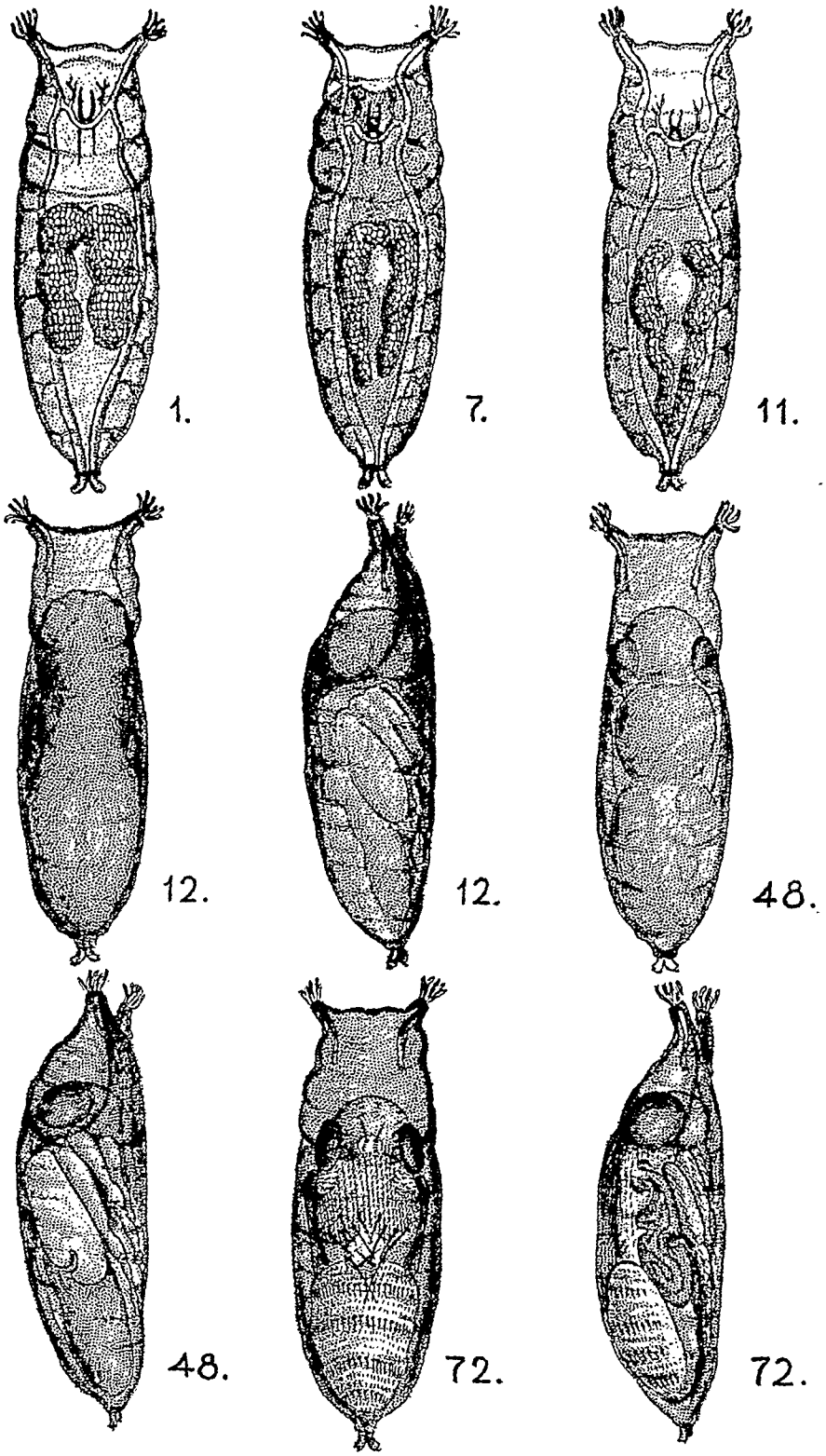


Fig. 1. Drawings showing stages during pupal (and prepupal) development which may be easily recognized with the unaided eye. The numbers indicate the age in hours after the collection of immotile white forms (*i.e.*, when maintained at 26° C.).

cent period, we shall, as a matter of convenience, refer to the cessation of movement as the beginning of pupation. All subsequent mention of pupal age, therefore, will be with reference to this stage as zero, unless otherwise indicated.

When the puparium is first formed it is white and translucent (Fig. 1, 1). Externally, the spiracles, two anterior and two posterior, are most conspicuous. Those in front are wide apart and branched, whereas those behind are close together and not branched. The anterior and posterior spiracles are connected through the body by two hollow trunks which are rendered clearly visible to the unaided eye by a combination of reflected and transmitted light. Beside the trunks, there are two other organs plainly visible within—the oral hooks (completely withdrawn) at the anterior end and the salivary glands located in the central region.

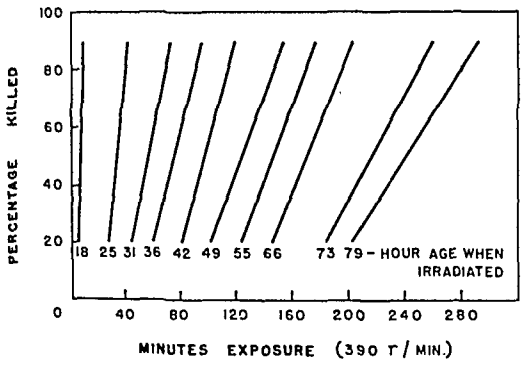


Fig. 2. Graph showing the percentage (20 to 90) of organisms killed as a function of exposure to radiation when treated at different ages. The curves for 2-, 6-, and 12-hour material were identical with the one for 18-hour material and are, therefore, not shown.

Within two hours after the cessation of movement, the organisms begin to turn brown and more opaque, a change which becomes complete within four hours. When the puparium is first formed, it is firmly attached to the tissues and organs beneath, but within three hours the structures in the anterior region begin to retract, causing a gas chamber to be formed. At about the same time, a second gas chamber appears in the central region. As these enlarge, the specific gravity of the organisms decreases so that they change from sinking to floating forms (Fig. 1, 7) at about seven hours, as determined by placing them in a vessel of water. Enlargement of the central gas chamber continues until its diameter occupies about 80 per cent of that of the entire organism. This stage is reached at about 11 hours (Fig. 1, 11).

Although the appendage primordia and the head anlage are well established at 11 hours, the organism appears headless and ill defined. However, within a 15-minute period beginning at approximately 11½ hours, it changes into a form with clearly defined head and body regions and with appendage rudiments. This takes place mainly through the collapse of the central gas chamber, eversion of the head and appendage primordia, and constrictions which set off the head, thorax, and abdomen (Fig. 1, 12).

After the elaboration of body parts at 11½ to 12 hours, no other externally visible change occurs until the organisms reach 48 hours of age (Fig. 1, 48). At this time red pigment begins to show in the eyes, the color becoming more intense during the following hours. The next distinctive change occurs at 72 hours when black pigment appears in the bristles, legs, and wings (Fig. 1, 72). Emergence of the imago from the puparium occurs at 96 hours.

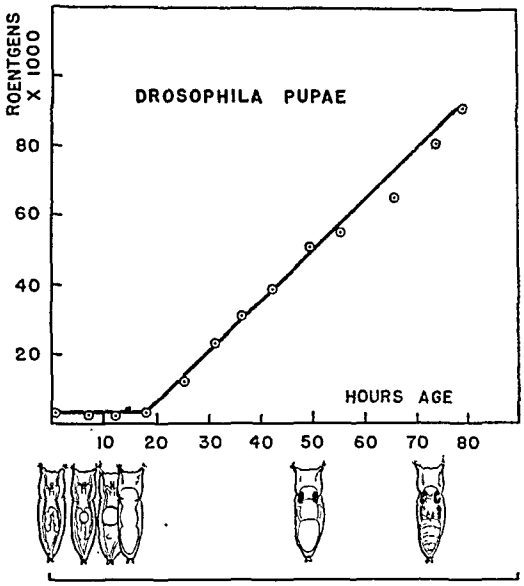


Fig. 3. Curve showing the number of roentgens (measured in air) required to kill 50 per cent of the organisms in a sample at different stages in development. The diagrams beneath show the distinguishing features at different stages.

There are, therefore, a number of easily recognizable stages manifest during the pupal period which may be used to designate the degree of development. These, together with the approximate times of occurrence (*i.e.*, when maintained at 26° C.), may be listed as follows:

STAGE	HOURS
1. Motility ceases	0
2. Browning begins	2
3. Organism will float on water	7
4. Delimitation of body regions	11½
5. Appearance of red eye pigment	48
6. Appearance of black pigment in bristles, legs, and wings	72
7. Emergence	96

2. *Changes in Radiosensitivity with Age.*—Investigation of the changes in radiosensitivity with age was undertaken with two points in mind: (1) determina-

tion of the suitability of *Drosophila* pupæ as test material for quantitative radiobiologic work, and (2) correlation of the changes in susceptibility to radiation with changes in the character of the developmental activity. Only the first point, however, will be dealt with in this section.

In order to correlate radiosensitivity and age in a significant way, it was necessary to restrict the age of individuals in the samples at various stages to fairly narrow limits. This was done by making use of the information given in Section 1 that the organisms are white for only about two hours after motility ceases. The procedure, therefore, was to collect immotile white forms and keep them in moist chambers at 26° C. until they reached the stage to be investigated.

Since the organisms are immotile and white for such a small portion of their life span, only a few will be found in this stage even in heavily populated cultures, thus making collection a real task. For this reason, the first experiments were performed with only two organisms per sample. By this means it was possible to ascertain the approximate radiosensitivity at different stages with a minimum of effort.

The samples were prepared by removing white pupæ from the walls of culture bottles with a soft wet brush and placing these on small squares of moist filter paper. When they had reached the age to be tested, the samples were arranged on a thin celluloid tray and taken to the x-ray machine for treatment.

The x-ray equipment used consisted of a Coolidge type, tungsten target, water-cooled tube operated at 200 kv. and 30 ma. At 33 cm. distance from the center of the target and with no filter, this equipment delivered 390 r min. (measured in air with a small spherical celluloid ionization chamber).

By using emergence as an end point, it was soon found that for the first 18 to 20 hours of pupal life the radiosensitivity changes little, if any, but that following this period it increases continuously until the end of development—thus confirming, in a general way, the observations of Mavor.

Having thus obtained an impression of the doses of radiation required at the different stages, more precise experiments were designed. The improvements, in the main, consisted of an increase in the number of organisms per sample and a clearer definition of the end point. Instead of two organisms per sample being used as before, 50 were employed and the killing as an end point was restricted to death at a particular stage. In case of the preliminary experiments, it was found that most of the pupæ which failed to emerge as imagos developed to the point of having well-defined red eyes and black bristles, legs, and wings. Thus, from the chart in Section 1 it appeared that development was fairly normal up to the point of emergence; it seemed as though the organs having to do with emergence had been injured or destroyed by the radiation (perhaps the glands which secrete the enzyme that weakens the puparium prior to the escape of the imago). Interestingly, this happens irrespective of when the pupa is irradiated in its life cycle (and even when irradiated during the larval stage).

Occasionally, however, in the preliminary experiments, it was found that some of the pupæ which failed to hatch did not display pigment and were of an even yellow color, thus indicating that death had occurred at an earlier stage in development. Investigation of this disclosed that if the dose of radiation is adequate, the pupæ may be killed at earlier stages, and, further, that usually about 1 or 2 per cent of the organisms in a sample die at the early stage whether irradiated or not. Thus, since we wished to obtain as uniform a response to the radiation as possible, it was decided to exclude arbitrarily from consideration all organisms which did not develop to the stage of black pigment in bristles, legs, and wings. This greatly improved the regularity of the experimental data because it restricted the ef-

fective dosage to what amounted to a threshold killing dose.

Four experiments were performed on material at the following ages: 2, 6, 12, 18, 25, 31, 36, 42, 49, 55, 66, 73, and 79 hours (*i.e.*, from the time white immotile pupæ were collected).

The data obtained were plotted to show how the percentage of organisms killed varied with the dose of radiation applied. It was thus found that for the range between 20 and 90 per cent killing, the experimental points could be fitted satisfactorily with straight line curves. The curves obtained in this manner for various ages of material and based on four separate experiments are shown in Figure 2. (The curve for 18-hour material also represents that for 2, 6, and 12 hours since the results obtained were identical in all these cases.)

It will be seen that beyond 18 hours the threshold effective dose becomes larger and that the range between the threshold and complete killing dose is increased. In order to obtain a clearer idea of how the changes in radiosensitivity occur, values were read from the curves at the 50 per cent level and plotted as shown in Figure 3. In this figure the curve appears to be flat for the first 18 hours, at which point it abruptly turns upward at an angle and again follows a straight line course. This means that the organisms do not change in susceptibility appreciably during the first 18 hours of pupal development, but that beyond this age they increase steadily in resistance. The significance of this interesting curve will be dealt with in the next section.

3. *Relation between Radiosensitivity and Developmental Activity.*—Having determined the changes in radiosensitivity with age, it became of interest radiobiologically to correlate the changes in susceptibility with the varied developmental activities at the different stages in order to ascertain whether or not any significant relation existed between them.

Since the larva is essentially a feeding form, it elaborates an extensive musculature for movement, highly specialized alimentary tract and gland system for the assimilation of food, and a complicated tracheal system for respiration. Thus, since, in general, these are of little or no use to the pupa or mature fly, one of the first functions of the organism following puparium formation is the removal of many of the larval structures. This takes place during the first 18 to 24 hours, mainly by histolysis, phagocytosis, and the casting off of structures. These functions constitute the major portion of the developmental activity during this period. The larval musculature disappears almost completely, the main portion of the digestive system is replaced, and the tracheal system is greatly modified.

From 24 to 48 hours, the organism is in a stage of relative inactivity. During this period there is little destructive activity and likewise little organogenesis. Active organogenesis, however, begins at about 48 hours and proceeds at a rapid rate until the imago emerges.

Three quite distinct stages are, therefore, recognized: the first, from 0 to 24 hours, which is characterized largely by destructive changes; the second, from 24 to 48 hours, by quiescence, and the third from 48 to 96 hours, by constructive changes.

In order to obtain a more tangible idea of the growth activity and at the same time some quantitative information on the developmental activity, it was decided to measure the rate of oxygen uptake at the different stages. For this a Warburg type of microrespirometer was used.

Samples consisting of 10 white pupæ each were placed in several different vessels and determinations were made daily. Each daily determination consisted of a series of four readings taken at 30-minute intervals. Since we were concerned only with relative respiratory rates at the different ages rather than the actual amounts of oxygen consumed and since the volumes of the vessels used varied less than 2 per cent, our results have been expressed in terms of millimeters change

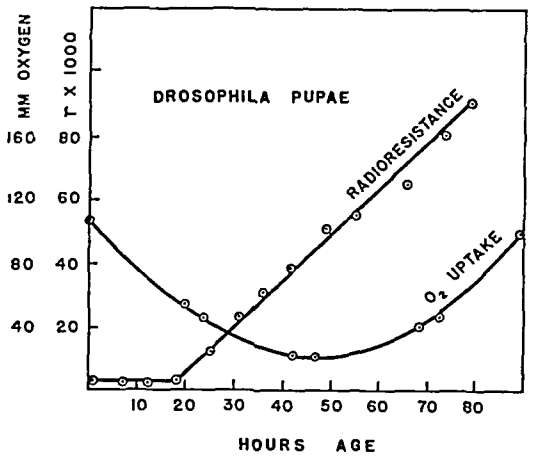


Fig. 4. Curves showing the relationship between oxygen uptake and radioresistance at different ages (pupæ only).

problem under consideration. The averaged values are plotted together with the results for changes in radiosensitivity (Fig. 4).

It will be seen that the rate of oxygen consumption drops gradually during the first 40 to 50 hours, after which it rises again. Because of the distinct downward trend of the curve for oxygen consumption at the beginning, it seemed pertinent to study its course during larval development. Accordingly, it was decided to extend the curves for both oxygen consumption and radiosensitivity over the entire life cycle of *Drosophila*. This was done in accord with methods already

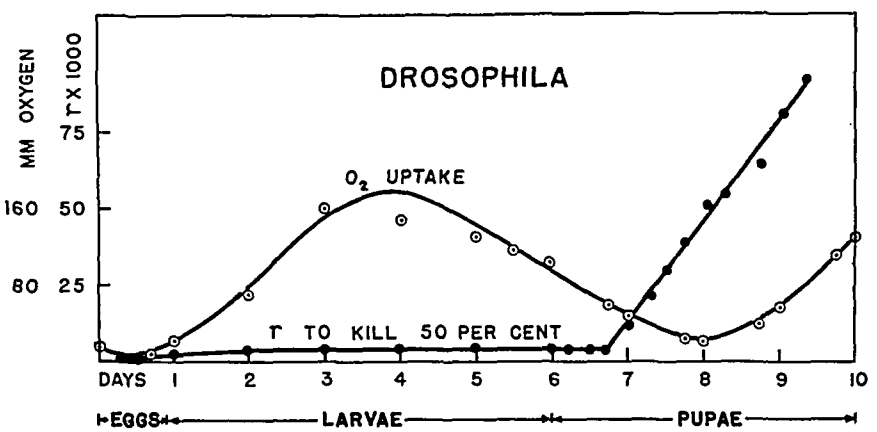


Fig. 5. Curves showing the relationship between oxygen uptake and radioresistance at different ages (entire life cycle).

in meniscus level of Brodie's fluid in the manometers, the readings, of course, receiving the customary thermobarometric corrections.

The values for changes in level of meniscus were plotted as a function of time and the best straight line drawn among the points. Readings were then taken from the curve for a period of 75 minutes respiration, this interval being chosen arbitrarily but always kept the same. After a series of exploratory tests, four such experiments were carried out and the results averaged. No table of values is being presented since a statistical evaluation of the data is not essential for the described and the results are given in Figure 5.

Figure 5 shows that the radiosensitivity changes little during larval development and that it is essentially the same as that for the early pupal stage. The rate of oxygen uptake, on the other hand, follows a varied course which may be correlated in a general way with the developmental activity. As the larva feeds and increases in size, its respiratory rate per organism increases, but with the approach of larval quiescence, this rate begins to fall. During the late larval and the early pupal stages, the fall continues at a rapid rate. However, when the destructive

changes slow down, the decline in rate of oxygen uptake likewise tapers off and as active organogenesis sets in there is a corresponding increase in oxygen uptake. The curve showing the rate of oxygen uptake may, therefore, be taken as a graphic representation of the developmental activity.

Examination of the two curves (Figs. 4 and 5) shows what appears to be a complete lack of positive correlation between rate of oxygen uptake and radiosensitivity.

4. *Reproducibility of Quantitative Responses to Radiation.*—Since we are concerned in this report with indicating the reliability of *Drosophila* pupæ as test material for radiobiologic work, it is necessary to indicate the precision of results obtainable.

It was indicated above in Section 2 and in Figures 4 and 5 that the radiosensitivity does not change appreciably during the first 18 hours of pupal life and that the susceptibility remains quite constant during this time. This long period of unchanging susceptibility appeared especially favorable for various types of experimentation and was accordingly investigated for precision of response.

The procedure used to limit the age of test material for this purpose was as follows: Active cultures of flies were maintained by seeding several bottles daily. To collect young pupæ, several such culture bottles containing many larvæ and a few pupæ were selected and the inner walls scraped free of pupæ at about 5 P.M. These bottles were then placed in the incubator overnight to allow other larvæ to crawl up the sides and change into pupæ. At 9 A.M. the newly formed pupæ were carefully removed with a soft wet brush and placed in a small vessel of water. Gentle stirring washed food particles free and allowed the larvæ and a few of the youngest pupæ to settle to the bottom. The floating pupæ were then removed to small moistened filter paper squares in a moist chamber and arranged side by side in rows (the latter to facilitate counting). The age limit of test material at the time of collection was, therefore, 7 to 16 hours since floating begins at seven hours (Section 1) and since the interval from 5 P.M. to 9 A.M. is 16 hours. There was no important reason for eliminating the pupæ less than seven hours old but these organisms dropped out in the washing procedure employed, and, since material was plentiful, no attempt was made to recover them. The samples as prepared for treatment consisted of 50 pupæ each.

Irradiation, which was carried out under precisely the same conditions as described in Section 2, was completed within one hour following collection and, therefore, occurred safely within the period of consistent radiosensitivity. Determination of the effect was, likewise, performed as described in Section 2.

The results for 17 consecutive experiments are shown in Table I. The values given represent the percentage killed in each sample of 50. Averages are shown at the bottom of each column together with the Standard Error of the Means. The averages and the limits of error have been indicated in the graph (Fig. 6).

TABLE I.—PERCENTAGE KILLED

Min. Expo.	0	5	6	7	8	9	10
1	2	7	26	63	94	100	100
2	0	8	20	62	82	100	100
3	0	10	21	28	71	100	100
4	0	1	19	38	78	95	100
5	2	10	27	52	89	100	100
6	4	12	21	43	77	98	100
7	0	6	16	47	71	100	100
8	0	3	26	44	75	95	100
9	3	10	21	46	83	99	100
10	3	12	15	52	86	97	100
11	2	11	18	36	59	95	100
12	3	13	28	57	88	98	98
13	2	7	32	52	84	100	100
14	5	8	35	41	86	95	98
15	4	12	33	50	70	98	100
16	0	14	46	58	90	98	100
17	5	12	29	67	85	100	100
Av.	2.6	9.2	25.5	49.2	80.9	98.1	100.0
σ_m	0.47	0.84	1.88	2.45	2.06	0.44	0.05

Examination of Figure 6 discloses (1) that the fertility of the controls is high; (2) that the reproducibility of the results is within limits satisfactory for quantita-

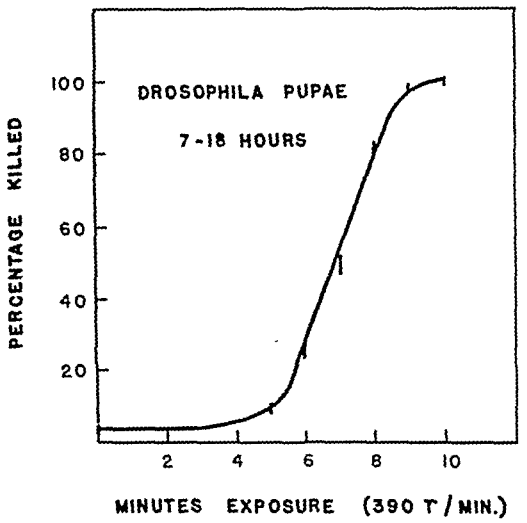


Fig. 6. Curve showing the percentage killed as a function of exposure to radiation, the vertical distance of the plotted points indicating the standard error of the mean.

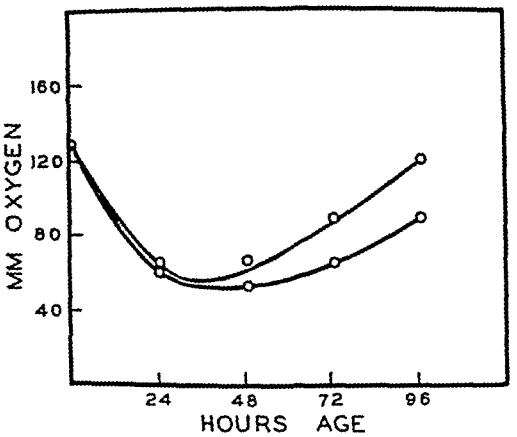


Fig. 7. Curves showing the rate of oxygen uptake during the pupal period for non-irradiated material and for material which received a lethal dose of radiation (5,000 r) when at the stage of white pupæ.

tive work; (3) that a distinct threshold effect is manifest, and (4) that the curve is of the sigmoid type.[3]

5. *Influence of a Lethal Dose of X-rays on Respiratory Rate.*—As a supplement to the measurements of respiratory rate in Section 3, similar measurements were made on organisms which had received lethal doses of radiation. This was done in an attempt to ascertain whether or not the respiratory activity is disturbed during development when a lethal dose of radiation is given early in the pupal period and death occurs late in this period.

For this work the dose of 5,000 r was used, throughout. This dosage was chosen for two reasons: first, because it is safely beyond the dose required to prevent 100 per cent of the organisms in a sample from hatching (Fig. 6), and second, because it was found in exploratory experiments that this dose does not prevent development from reaching the stage of black pigmented bristles, legs, and wings (72 hours, Section 1). The treatment, therefore, produced the same end points dealt with above and at the same time insured 100 per cent lethal effects in the samples.

The procedure used in collecting material, measuring the rate of oxygen uptake, and analyzing the results was the same in all essential details as described in Section 3. Six separate experiments were carried out and the averaged results have been plotted in Figure 6.

It will be seen that so far as these data are concerned the rate of oxygen uptake remains about the same during the first 20 to 30 hours but that it drops below and behind that of the non-irradiated material beyond this point.

It would appear from this that the radiation does, to some extent, affect the respiratory activity. Although completely satisfactory proof cannot be brought forth to the contrary, two objections may be raised at the outset against such an interpretation. First, it was observed in connection with the work described above (Sections 2 and 3 in which sub-lethal doses were administered) that emergence of the imago was delayed by several hours (6 to 10 in some cases). Going farther, it is reasonable to expect that for larger doses even greater delay would be produced. In accordance with this, it may be pointed out that the level of respiratory rate

[3] It will be noted that the curve presented here indicates that the organisms are somewhat more sensitive than does Müller's curve. This is because Müller's organisms varied in age from seven to 24 hours, his experiments being carried out before the radio-sensitivity-age factor was fully understood.

reached by the controls, say at 38 hours, is not reached by the irradiated until 64 hours—a delay of 22 hours; or that the level reached by the controls at 72 hours is not reached by the irradiated until 96 hours—a delay of 24 hours. From this it would seem that the delay in progress of the respiratory activity might be attributed to slower development.

Second, in addition to delay in attainment of levels of respiratory activity, the rate for the irradiated forms drops to a lower minimum at from 40 to 50 hours. This drop indicates that the respiratory rate per individual after receiving the lethal dose of radiation actually decreases. One cannot say, however, that this is due to the action of radiation on the respiratory apparatus since the findings thus far do not yield information as to the number of cells functional in the two cases at corresponding ages. Such information is needed before conclusions can be drawn.

The results presented here, therefore, lead to the presumption that although the respiratory activity may be altered somewhat, it is not by this means that the radiation exerts its lethal influence.

SUMMARY

1. The use of *Drosophila* pupæ as test objects for quantitative radiobiological work has been considered and investigated. Further, as a part of this investigation and as a supplement to it, the relation between radiosensitivity, developmental activity, and respiratory rate at various stages during the pupal period has been determined.

2. Using as a criterion of effect, development to the stage of pigmented bristles, legs, and wings, and failure of the imago to emerge from the puparium, it was found that during the first 18 hours the organisms maintain a strikingly uniform radiosensitivity, but that beyond this point their resistance increases at a rapid rate.

3. Considering normal development alone, three fairly distinct phases were recognized: the first, from 0 to 24 hours, characterized mainly by destruction and removal of larval organs; the second, from 24 to 48 hours, by relative quiescence, and third, 48 to 96 hours, by active organogenesis.

4. It was found that the rate of oxygen uptake in these forms decreases during the first phase, reaches a minimum during the second, and increases during the third, reaching a level at the time of emergence about equal to that at the beginning of the pupal stage.

5. There was, therefore, a positive correlation between the rate of oxygen uptake and developmental activity, but none between either of these and radiosensitivity.

6. The reproducibility of the quantitative response of pupæ 7 to 18 hours old to radiation was tested and found to be within narrow limits.

7. The general shape of the curve showing rate of oxygen uptake during the pupal period was not changed appreciably when lethal doses of radiation were administered early in the period. Only a slight delay was manifest which probably has little significance.

CONCLUSIONS

Drosophila pupæ may be recommended as favorable test material for radiobiologic studies for the following reasons: (1) they are easy to obtain in all seasons; (2) they are small, having a diameter of about 1 mm. and a length of about 2.5 mm.; (3) they are resistant to drying; (4) their susceptibility to radiation remains nearly constant for a period of about 18 hours; (5) they are relatively radiosensitive, 50 per cent being killed by a dose of 2,700 r, and (6) they yield results which can be reproduced within narrow limits from day to day.

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ROENTGENOGRAPHIC UNSHARPNESS OF THE SHADOW OF A MOVING OBJECT—II[1,2]

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INTRODUCTION

THIS is the purpose of this paper to present certain analytic and experimental results which have been obtained since the first paper[1] on this subject was published. These results are discussed briefly in three sections:

V.[3] Additional analytic data of the variation of exposure with distance along the unsharp shadow border when the intensity of the x-ray beam is constant.

VI. Tests by Dr. G. S. Pesquera[4] of the observed unsharpness of the shadow of a moving plane object; the technics for these tests were equivalent to modern chest roentgenographic technics.

VII. Analytic and experimental results for a moving plane object with an x-ray beam the intensity of which varies exponentially with time. The test films were made by R. B. Wilsey.[5]

A short description of the symbols used in this paper and in the first paper[1] follows. Figure 1 shows the arrangement of the film F, the moving plane object O, and the *effective* focal spot f; linear dimensions are in centimeters. The following quantities are used in the analysis; the x's are measured from x_0 upward:

$$x_1 = \frac{bf}{a} \qquad x_2 = \frac{a+b}{a} vT$$

$$vt_1 = \frac{bf}{a+b} \qquad x_3 - x_2 = x_1 = \frac{bf}{a}.$$

a = distance (cm.) from focal spot to object
b = distance (cm.) from object to film
f = length (cm.) of one side of square effective focal spot
v = velocity (cm./sec.) of object edge, parallel to film
T = exposure time (sec.)
x_1 = geometric unsharpness (cm.)
x_2 = unsharpness due to motion (cm.)
x_3 = "total unsharpness" (cm.).

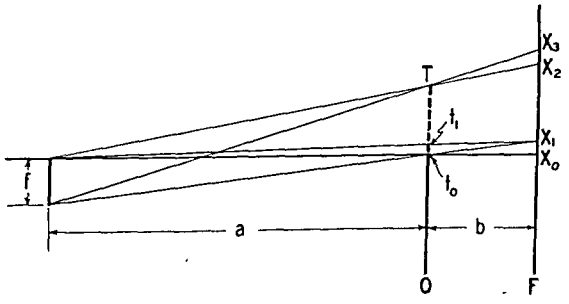


Fig. 1.[6] Schematic diagram *not drawn to scale* of the trace f of the effective focal spot of a roentgen-ray tube, the trace O of a plane object which moves with constant velocity v cm. per second in the direction t_0T, and the trace F of a plane x-ray film. A perspective view of the experimental apparatus is shown in Figure 5 of the first paper.[1]

The exposure, E, at any point on the film was calculated by integrating the instantaneous x-ray intensity at that point from $t = 0$ to $t = T$. The intensity of the beam at points outside of the shadow of the object was called I; the intensity of the beam at points *within* the shadow of the object was called I_0. Expressions for the exposure as a function of x were calculated

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[1] The first paper with this title was published in RADIOLOGY, 28, 450–465, April, 1937.

[2] Accepted for publication in August, 1939.

[3] The last section of the paper listed in Footnote 1 is numbered IV.

[4] Of the Metropolitan Life Insurance Company Sanatorium, Mt. McGregor, N. Y.

[5] Of the Research Laboratories of the Eastman Kodak Company, Rochester, N. Y.

[6] This figure is from the first paper.

(α, β, δ are values of x within ranges defined below):

$$E_\alpha = \frac{a^2(I - I_0)}{2bfv(a + b)}\alpha^2 + I_0T \qquad \left(0 \leqslant \alpha \leqslant \frac{bf}{a}\right)$$

$$E_\beta = \frac{a(I - I_0)}{v(a + b)}\beta + \frac{bf(I - I_0)}{2v(a + b)} + I_0T \qquad (0 \leqslant \beta \leqslant x_2 - x_1)$$

$$E_\delta = -\frac{a^2(I - I_0)}{2bfv(a + b)}\delta^2 + \frac{a(I - I_0)}{v(a + b)}\delta - \frac{bf(I - I_0)}{2v(a + b)} + IT \qquad (0 \leqslant \delta \leqslant x_3 - x_2).$$

V. ADDITIONAL ANALYTIC DATA FOR CONSTANT INTENSITY X-RAY BEAM

The formulas listed above for E_α, E_β, and E_δ were derived in terms of the arrangement shown in Figure 1. In this figure the object edge is at t_0 at the beginning of the exposure; the object extends *downward* from t_0 at this instant; the object moves upward during the exposure. In the experimental apparatus (see Fig. 5 of first paper[1]) the object edge is at t_0 at the beginning of the exposure; the object extends *upward* from t_0 at this instant; the object moves upward during the exposure. No explicit proof was given in the first paper that these two arrangements produce the same results. Such a proof follows.

When the object O is *below* t_0 at the beginning of the exposure, the exposure at any point from x_0 to x_3 may be calculated by means of one of the three expressions for E_α, E_β, E_δ given above. When the object is *above* t_0 at the beginning of the exposure (as it was in the experimental set-up) the exposure from $x = x_0$ to $x = x_1$ may be obtained by changing the expression for E_α in two ways:

1. Interchange I and I_0, because the object covers those parts of the film formerly uncovered, and *vice versa*.
2. Make the substitution $\alpha = -\left(\delta' - \frac{bf}{a}\right)$, because the range of α in the first case is the low density edge of the shadow; in the case now under consideration the x_0 edge of the unsharpness is the high density edge; finally, this transformation of α to a new variable δ' provides that δ' be zero at x_1 and $\frac{bf}{a}$ at x_0.

The result obtained by means of these operations may be written:

E_α (with I and I_0 interchanged, and with α replaced by $-\left(\delta' - \frac{bf}{a}\right)$

$$= -\frac{a^2(I - I_0)}{2bfv(a + b)}\delta'^2 + \frac{a(I - I_0)}{v(a + b)}\delta' - \frac{bf(I - I_0)}{2v(a + b)} + IT \qquad \left(0 \leqslant \delta' \leqslant \frac{bf}{a}\right)$$

This is precisely the expression for E_δ. Therefore, the values of E from x_2 to x_3 when the object starts from a position *below* the t_0 line are equal to the values of E from x_1 to x_0 when the object starts from a position *above* the t_0 line. Similar transformations show that: (1) The values of E from x_1 to x_2 when the object starts from a position *below* the t_0 line are equal to the values of E from x_2 to x_1 when the object starts from a position *above* the t_0 line and, (2) the values of E from x_0 to x_1 when the object starts from a position *below* the t_0 line are equal to the values of E from x_3 to x_2 when the object starts from a position *above* the t_0 line.

Therefore, the curves of exposure *versus* x measured from the less dense side of the unsharpness are the same, whether the object starts below the t_0 line and moves upward, or the object starts above the t_0 line and moves upward. The unsharpnesses of the two edges of an absorbing strip which moves across a film will be mirror images of each other; the curve of density *versus* x measured from the location of the mid-point of the object at time $T/2$ will be symmetrical about the line

TABLE I

Roentgen-ray Tube Voltage (kv. p.)	Roentgen-ray Tube Current (ma.)	Exposure Time (sec.)	Focal Spot-film Distance (ft.)	Side of Square Effective Focal Spot (mm.)	Velocity of Object for which Technic Produces Minimum Unsharpness (cm.-sec.)
70	150	1/10	5.5	1	1
70	150	1/20	4	1	2.5, 5.0
70	400	1/20	7	2	5.0
70	400	1/30	5.5	2	10.0, 15.0
70	400	1/60	4	2	15.0, 20.0

(Note that the technics are comparable to those used for chest roentgenography. The last column is discussed later in the paper.)

$x = 0$. This symmetry would not exist if the curve of x-ray intensity *vs.* time measured from the time $T/2$ were not symmetrical about $t = 0$.

The formulas listed above for E_α, E_β, and E_δ were calculated for the condition $\frac{x_2}{x_1} \geqslant 1$. If $\frac{x_2}{x_1} \leqslant 1$, a second set of formulas is required:

Constant Intensity Beam: $\frac{x_2}{x_1} \leqslant 1$

$$E^*_\alpha = \frac{a^2(I - I_0)}{2bfv(a + b)}\alpha^2 + I_0T \quad (0 \leqslant \alpha \leqslant x_2)$$

$$E^*_\beta = \frac{a(I - I_0)T}{bf}\beta + \frac{v(a + b)(I - I_0)T^2}{2bf} + I_0T \quad (0 \leqslant \beta \leqslant x_1 - x_2)$$

$$E^*_\delta = -\frac{a^2(I - I_0)}{2bfv(a + b)}\delta^2 + \frac{a(I - I_0)T}{bf}\delta - \frac{v(a + b)(I - I_0)T^2}{2bf} + IT \quad (0 \leqslant \delta \leqslant x_2).$$

These equations and those obtained by transformations like those described above for the E's when $\frac{x_2}{x_1} \geqslant 1$ show that the unsharpnesses on either side of the shadow of a moving absorbing strip are equal.

VI. TESTS OF UNSHARPNESS USING CHEST ROENTGENOGRAPHIC TECHNICS

Dr. G. S. Pesquera and his technical associate, Mr. E. C. Lasher, have constructed an instrument for making roentgenograms of pieces of lead which move parallel to the film with constant linear velocities. The lead strips are fastened to a disk of bakelite which is driven at constant angular velocity (one revolution per minute) by means of a motor, a speed-reducing gear train, and a belt; the edges of the six lead strips lie along a radius of the bakelite disk. The lead strips are so spaced, and the driving mechanism is so designed, that the linear velocities of the edges of the strips are 1.0, 2.5, 5.0, 10.0, 15.0, 20.0 millimeters per second. A photograph of this device is shown in a paper[7] recently published by Dr. Pesquera and Dr. Homer L. Sampson.

Dr. Pesquera has prepared several groups of films made with this device; he and several associates have visually compared the unsharpnesses of these films. He has sent the films and data to the author to be used in this paper. A short description of Dr. Pesquera's experiments and results follows.

The device described above was placed 20.3 cm. from the film. Five roentgenograms were made without intensifying screens by means of a four-valve apparatus with an impulse timer and a rotating anode tube using the technics shown in Table I.

For each exposure there were six unsharp shadow borders, for lead strips moving with linear velocities of 1.0, 2.5, 5.0, 10.0, 15.0, 20.0 mm. per sec. Thirty rectangular strips were cut from these films, each strip having a clear area (density about 0.14), an unsharp shadow of the moving edge of a lead strip, and a dark area (density about 1.0). These strips are like those shown in Figure 8 of the first paper. All strips which were the

[7] Pesquera, G. S., and Sampson, H. L.: The Evolution of Chest Roentgenographic Technic. Am. Jour. Roentgenol. and Rad. Ther., 40, 405, September, 1938.

roentgenograms of the piece of lead moving at 1.0 mm. per sec. were collected in one group; a second group comprised films of the lead strip moving with a velocity of 2.5 mm. per sec.; there were six groups of five films each. Each of seven observers was asked to examine each group of films, and to choose two or three films which, in his opinion, had the least unsharpness. The data of these observers are summarized in Table II. Bold face numbers in this table are identification numbers of film strips; numbers in parentheses are calculated observed unsharpnesses in mm.; a bold face identification number, and the corresponding technic, may be correlated by referring to the data listed with the same bold face number in Table III. *The data of Table II were recorded before the observed unsharpnesses were calculated.*

TABLE II

Object Velocity (cm. per sec.)	Film Strip Identification Number and Calculated Observed Unsharpness (mm.)	Votes of Seven Observers "most sharp of group"	"second most sharp of group"	"third most sharp of group"
1	**5** (0.13)	7	0	0
	12 (0.17)	0	5	0
	25 (0.18)	0	1	4
2.5	**11** (0.18)	7	0	0
	30 (0.19)	0	7	0
	4 (0.24)	0	0	3
	22 (0.23)	0	0	1
5.0	**8** (0.27)	4	3	0
	29 (0.25)	3	4	0
10.0	**23** (0.34)	5	2	0
	7 (0.50)	1	3	2
	26 (0.46)	0	2	1
	18 (0.34)	1	0	0
15.0	**20** (0.48)	4	3	0
	16 (0.36)	3	4	0
20.0	**17** (0.40)	7	0	0
	19 (0.63)	0	6	0

The data show that the seven observers' choices of the two or three most sharp roentgenograms of each group are in accord with determinations based upon calculations of the observed unsharpnesses, with two exceptions—film **18** and film **16**. The calculated observed unsharpnesses of these films are less than those of films chosen by the observers as more sharp. Two observers examined the two groups (v = 10.0 cm./sec. and v = 15.0 cm./sec.) again; the borders of films **18** and **16** appeared less sharp than other films of the same groups. Films **18** and **16** were examined by means of a low power (×2.5) microscope. The unsharp border appeared to comprise two narrow bands of high density perpendicular to the direction of motion of the lead strip; such striations were not perceptible in films **22, 4, 5, 24, 23.** It was concluded, therefore, that the two striations of films **18** and **16** were produced by the two pulsations of x-ray intensity of the 1/60 second exposure used for these films. *The observers' estimates of the unsharpnesses of films **16** and **18**, relative to the estimated unsharpnesses of other films for velocities of 10 cm. per sec. and 15 cm. per sec., are not in accord with the calculated unsharpnesses for films **16** and **18** because the intensity of the x-ray beam is not constant; the calculations are based upon the assumption of constant intensity.* The striations within the unsharp shadow border augment the appearance of unsharpness for exposures of 1/60 second; the effect is apparently negligible for exposures of 1/30 second or more for objects moving with velocities comparable to the velocities of objects within the chest. The apparent unsharpness of the shadow of a moving object may be greater for an exposure of 1/60 second than for some longer exposure. Therefore, exposures less than 1/30 second should not be used for objects having velocities from 5 cm./sec. to 15 cm./sec. if full wave rectified pulsating current flows in the roentgen-ray tube.

The values of the observed unsharpness listed in Tables II and III were calculated by means of four formulas:

$$U_0 = x_2 + x_1 \qquad 9 \leqslant \frac{x_1}{x_2} \leqslant \infty$$

$$U_0 = \frac{7}{16}\left(1 + \frac{1}{7}\frac{x_1}{x_2}\right)(x_2 + x_1)$$

$$1 \leqslant \frac{x_1}{x_2} \leqslant 9$$

$$U_0 = \frac{7}{16}\left(1 + \frac{1}{7}\frac{x_2}{x_1}\right)(x_2 + x_1)$$

$$1 \leqslant \frac{x_2}{x_1} \leqslant 9$$

$$U_0 = x_2 + x_1 \qquad 9 \leqslant \frac{x_2}{x_1} \leqslant 16.4$$

(16.4 is the highest value of x_2/x_1 in these data.)

The formulas differ from the formulas given in the original paper, which were:

$$U_0 = \left(1 - \frac{1}{2}\frac{x_2}{x_1}\right)(x_2 + x_1)$$

$$0 \leqslant \frac{x_2}{x_1} \leqslant 1$$

$$U_0 = \frac{1}{4}\left(1 + \frac{x_2}{x_1}\right)(x_2 + x_1)$$

$$1 \leqslant \frac{x_2}{x_1} \leqslant 3.$$

All of these formulas were chosen somewhat arbitrarily to correlate visual estimates of the relative unsharpnesses of test films; the formulas of the original paper[1] correlated the data described in that paper; the more extensive data now available are better correlated by the four formulas listed above and used to calculate the unsharpnesses of Table II and Table III. A brief description of the reasons for using these formulas follows:

The experimental films of Dr. Pesquera and Mr. Lasher have total unsharpnesses from 0.15 mm. to 2.41 mm.; the values of x_2/x_1 for the thirty exposures extend through the range $0.05 \leqslant \frac{x_2}{x_1} \leqslant 16.4$. Several films were held on the surface of a film illuminator, with a transparent scale (with divisions separated by 1 mm.), and examined by means of a low power (×2.5) microscope. The unsharpnesses (in millimeters) estimated in this manner were compared to the corresponding "observed unsharpnesses" calculated by means of the formulas of the original paper[1]:

$$U_0 = \left(1 - \frac{1}{2}\frac{x_2}{x_1}\right)(x_2 + x_1) \qquad 0 \leqslant \frac{x_2}{x_1} \leqslant 1$$

$$U_0 = \frac{1}{4}\left(1 + \frac{x_2}{x_1}\right)(x_2 + x_1) \qquad 1 \leqslant \frac{x_2}{x_1} \leqslant 3.$$

This comparison of visual estimates and calculated values shows that:

1. The calculated values and the visually estimated values of the "observed unsharpness" are approximately equal for the range $0.8 \leqslant \frac{x_2}{x_1} \leqslant 1.2$.
2. Visually estimated unsharpness is less than the unsharpness calculated by means of the two formulas above, when $\frac{x_2}{x_1} > 1.2$ and when $\frac{x_2}{x_1} < 0.8$.

Therefore, it was concluded that formulas might be chosen to correlate the experimental data more accurately than they are correlated by the formulas above. Examination of the films for which $\frac{x_2}{x_1} > 3$ shows that visually estimated unsharpness does not equal the calculated "total unsharpness" $(x_2 + x_1)$ until $\frac{x_2}{x_1} = 9$. This fact is clearly perceptible in Dr. Pesquera's films for which $(x_2 + x_1) < 2.5$ mm.; it is not demonstrated by the original test films[1] for which $(x_2 + x_1) = 10$ mm. Furthermore, the exposure formulas given in the first paper and in Section V show that the exposure is the same for any x whether $\frac{x_2}{x_1} = A (1 \leqslant A \leqslant 9)$ or $\frac{x_2}{x_1} = \frac{1}{A}$. The new formulas are chosen to take account of this fact. According to these new observations, therefore, the observed unsharpness may be more accurately calculated by means of the four formulas listed at the beginning of the last paragraph than by means of the formulas listed at the beginning of this paragraph.

VII. UNSHARPNESS OF THE SHADOW OF A MOVING OBJECT WITH X-RAY BEAM INTENSITY WHICH VARIES EXPONENTIALLY WITH TIME

The material described in the first paper[1] and in the first two sections of this paper is based upon the assumption that the intensity of the x-ray beam is constant during the exposure. It was shown in Section VI that the use of 1/60 second exposures with full wave rectified x-ray tube current and voltage produces results which are not in accord with the analysis because the x-ray intensity varies during the exposure. However, the data show that, for object velocities of the order of 5.0 cm./sec. to 15 cm./sec., the variations of intensity during exposures of 1/30 second or more do not produce a perceptible deviation from the analytic results. Finally, it was shown in Section V that intensity *vs.* time curves which are symmetrical about time $T/2$ produce equivalent unsharp shadow borders for both sides of a moving absorbing strip.

An example of an intensity *vs.* time curve which is not symmetrical about time $T/2$ is the output of a condenser-discharge roentgenographic apparatus. Such equipment has been used with discharge control apparatus of several types. Apparatus having mechanical switches for connecting the charged condenser to the x-ray tube have x-ray tube voltage and current wave forms which vary approximately exponentially with time. The wave forms

TABLE III

Focal spot-film distance (ft.)	4	5.5	7	4	5.5
Length of side of effective focal spot (mm.)	2	2	2	1	1
Exposure time (sec.)	1/60	1/30	1/20	1/20	1/10
Film Number	14	21	25	12	5
Linear velocity (mm./sec.)	1	1	1	1	1
x_2/x_1	0.05	0.14	0.26	0.30	0.82
U_0 (mm.)	0.42	0.27	0.18	0.17	0.13
Estimate of unsharpness (1 judged most sharp) S. R. W.	5	4	3	2	1
Estimate of unsharpness (1 judged most sharp) D. B. O'N.	5	4	3	2	1
Film Number	15	22	30	11	4
Linear velocity (mm./sec.)	2.5	2.5	2.5	2.5	2.5
x_2/x_1	0.13	0.34	0.66	0.75	2.06
U_0 (mm.)	0.43	0.23	0.19	0.18	0.24
Estimate of unsharpness (1 judged most sharp) S. R. W.	5	4	2	1	3
Estimate of unsharpness (1 judged most sharp) D. B. O'N.	5	4	2	1	3
Film Number	13	24	29	8	6
Linear velocity (mm./sec.)	5	5	5	5	5
x_2/x_1	0.25	0.68	1.31	1.50	4.12
U_0 (mm.)	0.34	0.25	0.25	0.27	0.49
Estimate of unsharpness (1 judged most sharp) S. R. W.	4	3	1	2	5
Estimate of unsharpness (1 judged most sharp) D. B. O'N.	4	3	2	1	5
Film Number	18	23	26	7	3
Linear velocity (mm./sec.)	10	10	10	10	10
x_2/x_1	0.50	1.36	2.60	3.00	8.20
U_0 (mm.)	0.34	0.34	0.46	0.50	1.21
Estimate of unsharpness (1 judged most sharp) S. R. W.	4	1	2	3	5
Estimate of unsharpness (1 judged most sharp) D. B. O'N.	4	1	2	3	5
Film Number	16	20	28	9	1
Linear velocity (mm./sec.)	15	15	15	15	15
x_2/x_1	0.75	2.04	4.00	4.50	12.4
U_0 (mm.)	0.36	0.48	0.71	0.85	1.85
Estimate of unsharpness (1 judged most sharp) S. R. W.	2	1	3	4	5
Estimate of unsharpness (1 judged most sharp) D. B. O'N.	2	1	3	4	5
Film Number	17	19	27	10	2
Linear velocity (mm./sec.)	20	20	20	20	20
x_2/x_1	1.00	2.73	5.20	6.00	16.4
U_0 (mm.)	0.40	0.63	1.00	1.14	2.41
Estimate of unsharpness (1 judged most sharp) S. R. W.	1	2	3	4	5
Estimate of unsharpness (1 judged most sharp) D. B. O'N.	1	2	4	3	5

for apparatus with electronic switches are also approximately exponential. In some condenser-discharge equipments the condenser is always connected to the x-ray tube and the exposure is produced by increasing quickly the temperature of the x-ray tube cathode; in others an inductance (a choke coil) is connected in series with the tube and condenser; the wave forms of voltage and current in these cases are not exponential, and they are different from each other. The discussion which follows is concerned solely with condenser-discharge apparatus in which the x-ray tube voltage and the x-ray tube current vary exponentially with time.

The exposure ratings of x-ray tubes used with condenser-discharge equipment have not been satisfactorily standardized. Some experiments suggest that tubes will safely withstand higher exposures (greater watt-seconds) when the energy is supplied by a charged condenser than they will withstand when the energy is supplied directly by means of a transformer and rectifier. Tube ratings often are based on the assumption that the energy of the exposure determines the rating, regardless of the kind of power-supply system. Because of this lack of agreement, and for other reasons stated below, no attempt is made in the following material to specify optimum exposure technics for condenser-discharge equipment.

It is the purpose of this Section (VII) to show that the methods described in Sections I to VI may be applied to exposure technics for which the x-ray intensity varies exponentially with time. It may be inferred from the results of this Section that the general methods of calculating unsharpness may be applied to any form of intensity *vs.* time variation; such calculations may be complicated from the algebraic point of view but they are not likely to be perplexing from the theoretical point of view.

The equations for calculating the ex-

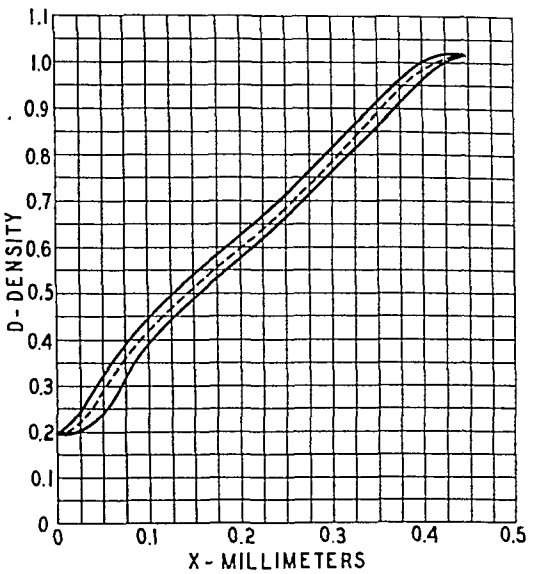


Fig. 11.

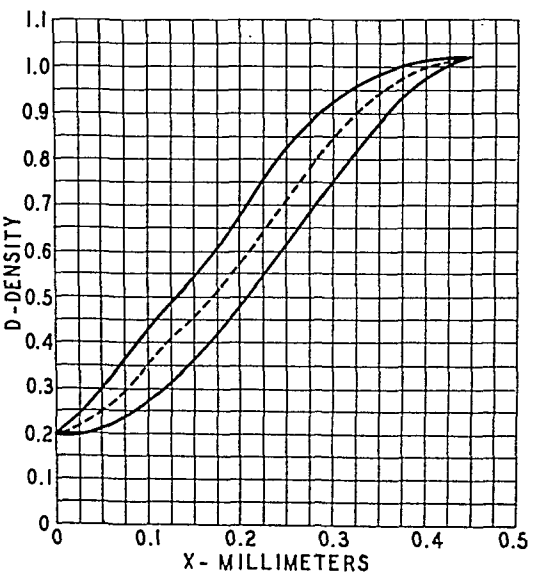


Fig. 12.

Fig. 11. Calculated variations of roentgenographic densities D with x (Fig. 1) for the leading and trailing edges of a moving absorbing strip: (1) Top curve—exponential variation of intensity with exposure; $x_2/x_1 = 0.2$; curve for leading edge of absorbing strip. (2) Bottom curve—exponential variation of intensity with exposure; $x_2/x_1 = 0.2$; curve for trailing edge of absorbing strip. (3) Dotted curve—constant intensity during exposure; $x_2/x_1 = 0.2$; curve for either leading edge or trailing edge of absorbing strip.

Fig. 12. Calculated variations of roentgenographic densities D with x (Fig. 1) for the leading and trailing edges of a moving absorbing strip: (1) Top curve—exponential variation of intensity with exposure; $x_2/x_1 = 0.8$; curve for leading edge of absorbing strip. (2) Bottom curve—exponential variation of intensity with exposure; $x_2/x_1 = 0.8$; curve for trailing edge of absorbing strip. (3) Dotted curve—constant intensity during exposure; $x_2/x_1 = 0.8$; curve for either leading edge or trailing edge of absorbing strip.

posure at any point from x_0 to x_3 in Figure 1 are derived as follows: The procedure is similar to that described in Section I of the first paper.[1] The x-ray intensity at the surface of the film is assumed to vary exponentially with time; the initial intensity at a point on the film "covered" by the object is I_0'; the initial intensity at a point on the film not "covered" by the object is I'; the roentgenographic exposure is assumed to end at the instant when the intensity has decreased to one-tenth of the initial intensity. Therefore:

$$i_0' = I_0'\epsilon^{-\frac{2.3t}{T}} \qquad i' = I'\epsilon^{-\frac{2.3t}{T}}$$

$i_0' = I_0'$ when $t = 0$

$i' = I'$ when $t = 0$

$i_0' = 0.1I_0'$ when $t = T$

$i' = 0.1I'$ when $t = T$.

The exposure at x_0 (Fig. 1) is:

$$E_{x_0}' = I_0'\int_0^T \epsilon^{-\frac{2.3t}{T}}\,dt = \frac{I_0'T}{2.3}(1 - 0.1) = \frac{I_0'T}{2.55}.$$

The exposure at x_3 is:

$$E_{x_3}' = I'\int_0^T \epsilon^{-\frac{2.3t}{T}}\,dt = \frac{I'T}{2.55}.$$

From this point the analysis proceeds in the same manner as that described in Section I. This analysis is straightforward, though somewhat tedious; the detailed description is therefore omitted. The expressions for calculating the exposure at any point from x_0 to x_3, for $\frac{x_2}{x_1} \geqslant 1$ are:

$$E_\alpha' = \frac{v(a+b)(I'-I_0')}{bfa_0^2}(\epsilon^{a_0t_x} - 1) - \frac{a(I'-I_0')}{bfa_0}\alpha + \frac{I_0'}{a_0}(\epsilon^{a_0T} - 1) \qquad (0 \leqslant \alpha \leqslant x_1)$$

where $a_0 \equiv \frac{-2.3}{T}$ and $t_\alpha = \frac{a\alpha}{v(a+b)}$

$$E_\beta' = \frac{v(a+b)(I'-I_0')}{bfa_0^2}(\epsilon^{a_0t_{\beta_2}} - \epsilon^{a_0t_{\beta_1}}) - \frac{I'}{a_0} + \frac{I_0'}{a_0}\epsilon^{a_0T} \qquad (0 \leqslant \beta \leqslant x_2 - x_1)$$

where $a_0 \equiv \frac{-2.3}{T}$ and $t_{\beta_2} = \frac{a(x_1+\beta)}{v(a+b)}$

and $t_{\beta_1} = \frac{a\beta}{v(a+b)}$

$$E_\delta' = \frac{v(a+b)(I'-I_0')}{bfa_0^2}(\epsilon^{a_0T} - \epsilon^{a_0t_\delta}) + \frac{a(I'-I_0')}{bfa_0}\epsilon^{a_0T}\delta - \frac{I'}{a_0} + \frac{I_0'}{a_0}\epsilon^{a_0T} \qquad (0 \leqslant \delta \leqslant x_1)$$

where $a_0 \equiv \frac{-2.3}{T}$ and $t_\delta = T - \frac{bf}{v(a+b)} + \frac{a\delta}{v(a+b)}$.

Expressions have been calculated for E_α', E_β', and E_δ' for cases in which $\frac{x_2}{x_1} \leqslant 1$. These equations are not listed here because it is the purpose of this Section to show the form of the results rather than the details of the results.

It was pointed out in Section V that an intensity *vs.* time curve which is not symmetrical about $T/2$ might be expected to produce dissimilar unsharpnesses of the shadow borders of a moving absorbing strip. The formulas for the exposures of points on the film for the case of the object placed *above* t_0 (Fig. 1) at the beginning of the exposure and moved upward during the exposure may be obtained by transforming the above equations for E_α', E_β', and E_δ' in a manner similar to that described in Section V. The results are not reproduced here, but their forms are illustrated in Figures 11, 12, 13, and 14 which are described below.

It is important to know what form of density *vs.* x curve is obtained from the formulas E_α', E_β', and E_δ', and film data like those described in Section II. Calculations were made for $(x_1 + x_2) = 0.45$ cm., using several values of x_2/x_1. Numerical values of I' and I_0' were so chosen that $E_{x_0}' = 0.15$ and $E_{x_3}' = 3$; data for

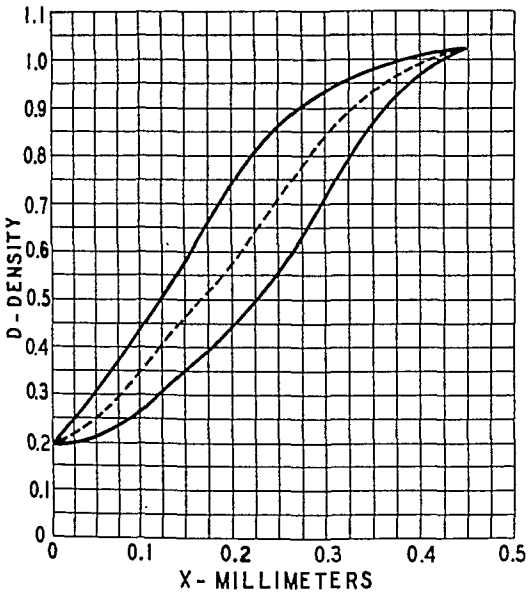


Fig. 13.

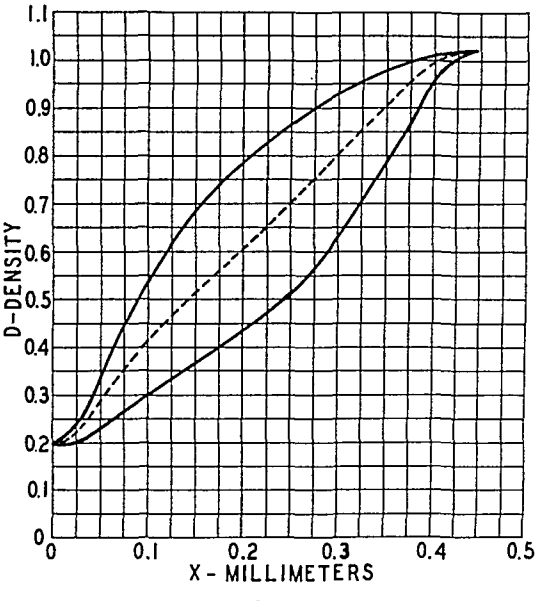


Fig. 14.

Fig. 13. Calculated variations of roentgenographic densities D with x (Fig. 1) for the leading and trailing edges of a moving absorbing strip: (1) Top curve—exponential variation of intensity with exposure; $x_2/x_1 = 1.5$; curve for leading edge of absorbing strip. (2) Bottom curve—exponential variation of intensity with exposure; $x_2/x_1 = 1.5$; curve for trailing edge of absorbing strip. (3) Dotted curve—constant intensity during exposure; $x_2/x_1 = 1.5$; curve for either leading edge or trailing edge of absorbing strip.

Fig. 14. Calculated variations of roentgenographic densities D with x (Fig. 1) for the leading and trailing edges of a moving absorbing strip: (1) Top curve—exponential variation of intensity with exposure; $x_2/x_1 = 4.0$; curve for leading edge of absorbing strip. (2) Bottom curve—exponential variation of intensity with exposure; $x_2/x_1 = 4.0$; curve for trailing edge of absorbing strip. (3) Dotted curve—constant intensity during exposure; $x_2/x_1 = 4.0$; curve for either leading edge or trailing edge of absorbing strip.

the film described in Section II indicate that these exposure values correspond to $D_{x_0} = 0.2$ and $D_{x_3} = 1.02$. Figures 11, 12, 13, 14 show graphically the results of these calculations for values of x_2/x_1 as follows: Figure 11, $x_2/x_1 = 0.2$; Figure 12, $x_2/x_1 = 0.8$; Figure 13, $x_2/x_1 = 1.5$; Figure 14, $x_2/x_1 = 4.0$. The top curve in each figure represents the change of density with x from x_0 upward for the conditions shown in Figure 1; the bottom curve in each figure represents the change of density with x from x_3 downward for the case when the object is *above* t_0 when $t = 0$; the dotted curve (between the top and bottom curves) represents the change of density with x for either original position of the object when the x-ray beam intensity is constant during the exposure.

These data point to the following conclusions:

1. The unsharp shadow borders of objects for which $x_2/x_1 \ll 1$ are nearly alike for constant intensity exposures and for exposures the intensities of which vary exponentially with time.
2. When $x_2/x_1 > 1$ the shadow borders of constant intensity exposures differ greatly from the borders of exponentially varying exposures. Furthermore the shadow

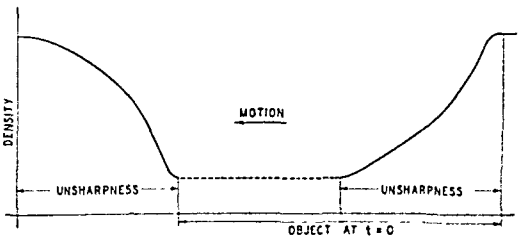


Fig. 15. Curve showing the estimated variation of density with distance along the shadow of a moving strip with condenser-discharge apparatus. Note that the shapes of the two curves of the unsharp shadow borders are quite similar to those shown in Figure 14. Figure 14 shows the calculated curves of the unsharpness for $x_2/x_1 = 4.0$; Figure 15 shows the visually estimated unsharpness curve for a roentgenogram made with condenser-discharge apparatus for $x_2/x_1 = 4.0$.

borders of opposite edges of an absorbing strip differ greatly if the intensity varies exponentially. Roentgenographic exposures of Pesquera's apparatus were made by Wilsey to test these conclusions.

The unsharp shadow borders of moving lead strips have the characteristics predicted by the analysis given above. If x_2/x_1 is less than about 0.5, the unsharpness appears to be a region of approximately uniform density gradient, from the point of minimum density to the point of maximum density; the unsharpnesses on either side of the shadow of the moving strip appear to be equivalent. If x_2/x_1 is greater than about 0.5, the shadows on either side of the moving strip differ greatly. For $x_2/x_1 = 4$ the shadow of the strip appears to have the form shown in Figure 15; this appearance corresponds to the analytic form of Figure 14.

SUMMARY

Additional experimental data and critical comments submitted to the author by other workers have been used to extend the scope of the analysis of the unsharpness of the shadow of a plane-absorbing object which moves with constant velocity parallel to the film during roentgenographic exposures. The following results are described briefly in terms of the analysis and experiments described in a paper[1] published in 1937:

1. The shadow of a moving absorbing strip is symmetrical about the center line of the shadow if the x-ray beam intensity is constant during exposure; in general, the shadow is symmetrical if the intensity *vs.* time curve is symmetrical about the time $T/2$.

2. Formulas are given for calculating the exposure (constant intensity) at any point in the area of unsharpness for $x_2/x_1 \leqslant 1$.

3. Tests by Pesquera suggest a modification of the formulas of U_0, presented in the first paper, to improve the accuracy of calculated observed unsharpnesses.

4. Pesquera submitted his test films to several experienced observers who were, however, unfamiliar with calculated unsharpnesses at the time they examined the films. Their observations confirm the analysis except for films made in 1/60 second at velocities greater than 5 cm./sec.; the intensity variations for such exposures preclude the use of the analytic formulas to predict the form of their unsharpnesses.

5. It is shown that a particular asymmetrical intensity *vs.* time curve that varies exponentially with time may be used as a basis for calculating the unsharpness for condenser-discharge roentgenographic apparatus. These calculations are found to be in accord with experimental data prepared by Wilsey, using condenser-discharge apparatus.

CASE REPORT

OSTEOGENIC SARCOMA OCCURRING IN FRAGILITAS OSSIUM[1]

A CASE REPORT

By F. C. JEWELL, M.D., *and* J. E. LOFSTROM, M.D., *Detroit, Michigan*

From the City of Detroit Receiving Hospital

The occurrence of osteogenic sarcoma and fragilitas ossium is not uncommon when alone, but a search through the literature failed to reveal a record of a case in which the two appeared together.

[1] Accepted for publication in May, 1939.

Lobstein, in 1833, first used the term "osteopsathyrosis" to denote idiopathic as well as other types of fragility of the bones. Looser later called it "osteogenesis imperfecta congenita" and "osteogenesis imperfecta tarda," the former being prenatal and affecting the bones of the skull and causing a large number of fractures of all the bones; and the latter being postnatal and affecting chiefly long bones. The pathologic picture was described as the same in both by Cinelli (1) and Ford (3).

The microscopic picture of fragilitas ossium shows a deficient functional activity of the osteoblasts. The preliminary processes of

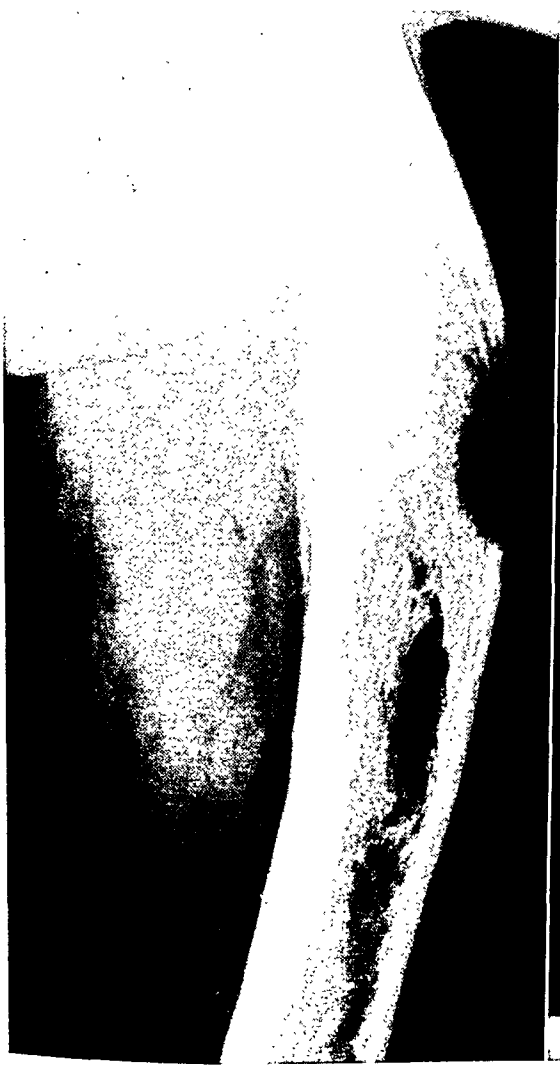


Fig. 1.

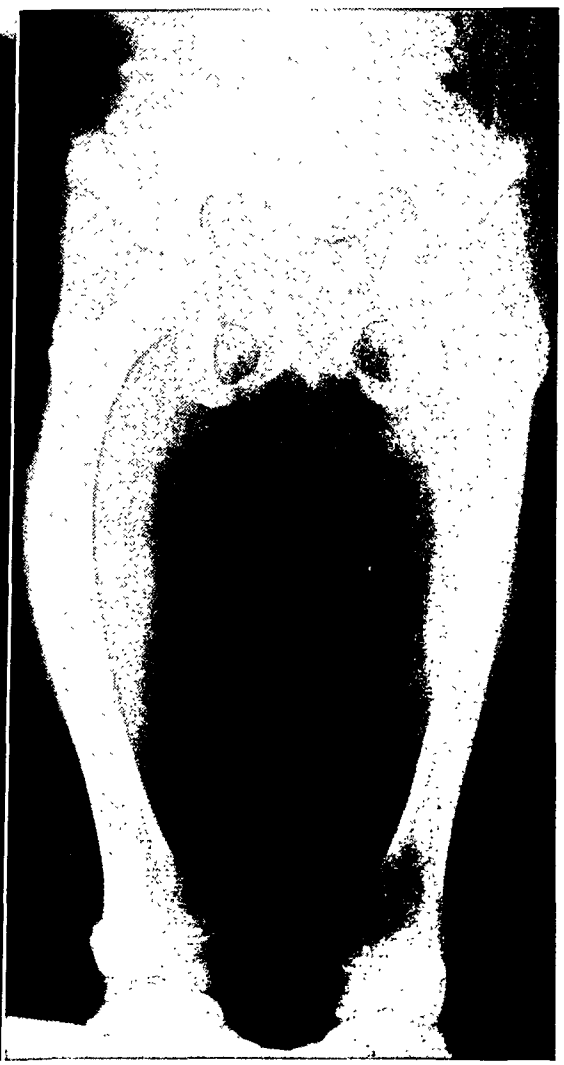


Fig. 2.

Fig. 1. Right femur showing old fracture and thinning of the cortex inferiorly.
Fig. 2. Femur of the patient's daughter, showing old fracture; increased caliber of medullary canals; deformity.



Fig. 3. Osteolytic process involving right ilium and upper half of acetabulum.

ossification are normal. There has been no general agreement as to any primary abnormality in the marrow or in the function of the osteoblasts. Metaplasia is frequent but probably compensatory, not primary (Bronson, 2). It is generally felt now, although we know nothing of the etiology, that osteogenesis imperfecta and fragilitas ossium are two separate pictures, the first being non-hereditary and the latter hereditary. There is no evidence to denote any change in the blood levels of phosphatase, chloride, phosphorus, or calcium in fragilitas ossium. The diagnosis of a case of fragilitas ossium showing an associated osteogenic sarcoma prompted us to make the following report.

Report of Case.—E. W., white male, 49 years of age, was admitted to the City of Detroit Receiving Hospital on Feb. 2, 1939, with the complaint of pain in the right hip. The patient stated that his present illness started in February, 1938, when he noted a dull, non-radiating pain localized in the region of the right hip. Symptoms were present on motion and from one to three hours after retiring. The condition had slowly progressed for the past year. Occasionally he was unable to work because of the condition of his hip. On Feb. 2, 1939, he fell on the ice, injuring his right hip. He was unable to rise and by virtue of his brittle bones he felt he had fractured his femur.

Past Illness.—At the ages of 7, 18, and 35 years the patient had fractured his right femur from minor trauma to the area (Fig. 1). At the age of 21, he had fractured his left femur. It is interesting to note that the patient's father had had his femora fractured several times and is now a cripple. A brother had had at least 12 fractures of the thigh and leg bones. One of the patient's daughters sustained two fractures of the right femur (Fig. 2). His grandmother suffered the same condition, and, in short, the brittle bones could be traced verbally for from 200 to 300 years in his family. The past history was otherwise irrelevant except that his teeth had become carious and removal had been necessary at the age of 18. He had always had blue sclera, and, from the age of 20, he had been slowly becoming deaf. There had been no familial history of carcinoma. His mother expired of pulmonary tuberculosis.

Physical Examination.—This revealed the patient to be well nourished and well developed. Skin, negative; head, negative; eyes, blue sclera; ears, otosclerotic deafness, both drums perforated; nose, negative; mouth, teeth removed; neck, negative; lymph glands, negative; heart and lungs, negative; vascular system, blood pressure, 130/85; abdomen, a hard, immovable mass in the right lower quadrant extending upward from the inguinal region a distance of 7 cm. The surface was smooth and attached to the underlying tissues. Bones, moderate tenderness of the right ilium 10 cm. from the symphysis. Some lateral bowing of the femora was noted.

Laboratory Examination.—Urine showed occasional white blood cells. Klein test was negative. Hemoglobin, 12.5 grams per cent; white blood cells, 8,500; red blood cells, 4.15 millions; filamentous neutrophils, 64 per cent; nonfilamentous neutrophils, 12 per cent; lymphocytes, 24 per cent. Blood phosphatase, 5.5 units; blood chloride, 462 mg./100 c.c.; serum phosphorus, 4.4 mg./100 c.c.; serum albumin, 3.5 per cent; serum globulin, 3.1 per cent; blood calcium, 9.4 mg.; sedimentation rate, 12 minutes.

Roentgenographic Findings.—The right femur (Fig. 1) reveals evidence of old healed fractures at the junction of the upper and middle thirds and extending to the region of the lesser trochanter. Union is complete with moderate lateral bowing. In addition, there is thinning of the femoral cortex especially on its lateral aspect throughout the entire length of the shaft. The medullary canal is widened. Similar findings were noted in the left femur. Roentgenograms of the skull, chest, thoraco-lumbar spine, and humerus showed no abnormalities.

The pelvis (Fig. 3) shows evidence of an area of osteolysis involving the right ilium and also the upper half of the acetabulum, with disruption of the articular surface. The area extends in an irregular manner superiorly to the vicinity of the anterior superior spine and also through the medial wall of the ilium. There are a few islets of bone remaining within the lytic area. There is no new bone production. There is a soft-tissue mass visible, extending laterally and also into the pelvic cavity. There is slight abnormality of the contour of



Fig. 4.

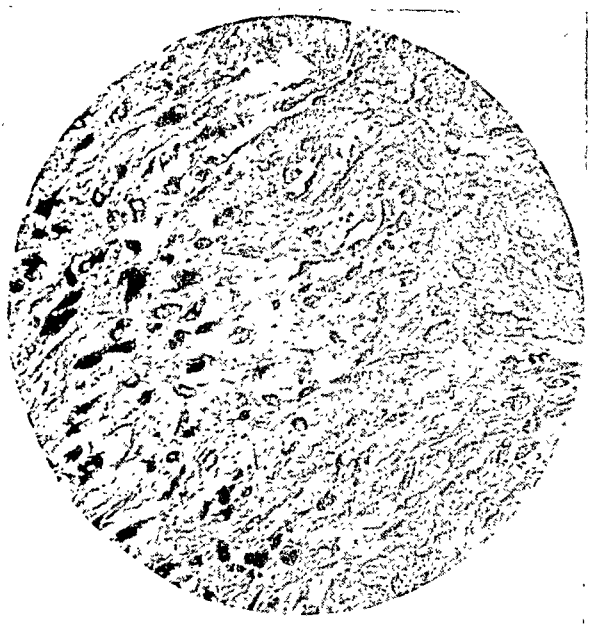


Fig. 5.

Fig. 4. Biopsy of neoplastic areas composed of spindle cells containing osteoid deposits with calcification of atypical trabeculæ (low power).

Fig. 5. Spindle-shaped neoplastic cells differentiated into osteoblasts (high power).

the head of the femur which is due to faulty weight-bearing secondary to fractures of the femur. The osseous structure is otherwise normal.

Roentgen examination shows moderate lateral bowing in the middle and upper thirds of the right femur of the patient's daughter (Fig. 2). The cortex in this instance is not noticeably thinned although the medullary canal is of increased caliber. There is resultant shortening of the shaft and slight widening of the distal end of the femur at the epiphysis. In view of the familial history and the findings presented by the father, it was considered that these were of the nature of fragilitas ossium.

It was felt that the process in the right ilium was due to an area of primary malignancy. Sarcoma, lymphoblastoma, and Ewing's tumor were considered in the differential diagnosis. The condition of the femora with associated fractures and familial history is relatively typical of fragilitas ossium.

On Feb. 21, 1939, a biopsy was taken from the mass over the right inguinal region. The report given by Dr. O. A. Brines, pathologist, is as follows (Figs. 4 and 5): "The sections represent a neoplasm composed chiefly of spindle-shaped cells embedded in an abundant mucinous stroma. In some areas the spindle neoplastic cells differentiate into osteoblasts, and small deposits of osteoid material have been formed which in some instances assume a definite trabecular pattern. In some of these trabeculæ calcification has occurred." Following this it was the decision of the Tumor Clinic that this patient should have a course of deep x-ray therapy.

SUMMARY

1. A case of associated fragilitas ossium and osteogenic sarcoma is presented.

2. Although these two conditions occurred together, it is not felt that there is any direct relationship between them.

3. The familial history of the fragilitas ossium can be traced verbally for two or three hundred years.

4. A review of the literature failed to reveal any similar relationship in the pathologic conditions.

REFERENCES

(1) CINELLI, A. A.: Fragilitas Ossium with Otosclerosis and Blue Sclera: Report of a Case. Arch. Otolaryng., 25, 309–311, March, 1937.

(2) BRONSON, E.: Fragilitas Ossium and its Association with Blue Sclera and Otosclerosis. Edinburgh Med. Jour., 18, 240–281, April, 1917.

(3) FORD, S.: Congenital Bone Dysplasia. RADIOLOGY, 30, 117–121, January, 1939.

THE WASHINGTON SCENE

Although the so-called National Health Bill drafted by Senator Wagner last year failed to reach the Senate floor, reports from some sources are that it is currently being revised and will be reintroduced before the Seventy-sixth Congress adjourns. Meanwhile, various bills intended to accomplish certain aims of the original bill have been introduced in the national Congress.

The Wagner-George Bill, *S. 3230*, upon which hearings have already been held, called for appropriation of $10,000,000 the first year for construction of hospitals in needy areas, title to remain in the Federal Government. The Mead Bill, *S. 3246*, proposed to authorize a federal appropriation of $300,000,000 for loans to States for hospitals, water, sewers, stream pollution control, and related projects and facilities. Of the total, $100,000,000 was to be devoted to hospital projects. Senator Mead, in his bill, defines a hospital as any institution for treating disease, including any health, diagnostic, or treatment center, station, or clinic.

A bill introduced by Senator Taft, of Ohio, on April 18, embodies certain of the recommendations contained in a memorandum left by representatives of the American Medical Association following a conference in Washington on January 10. It would provide that the Federal Government contribute, for a period of five years and from annual appropriations of $10,000,000, from 40 to 90 per cent of the cost of hospitals constructed with the approval of a National Advisory Hospital Council appointed by the Surgeon-General with the approval of the Federal Security Agency. Title to the hospitals would rest in the local jurisdiction.

In all these bills, equipment to be installed in the hospital is contemplated as a part of the cost of construction, it seems. In the case of the Wagner-George Bill, at least, this would entail a certain amount of governmental supervision in the use of this equipment. The status of radiologists operating in such hospitals is a matter for conjecture.

A revised version of the Wagner-George Bill was reported out for the Senate Committee on Education and Labor, by Senator James E. Murray, of Montana, on April 30. The Committee frankly states in its report that the new bill is designed to accomplish part of the original Wagner Health Bill and that it is only the initial step in the Committee's projected program.

The revised bill would appropriate $10,000,000 the first fiscal year to be used for hospital construction and an equal amount for each of the five succeeding years to be used for grants-in-aid to States and subdivisions thereof for construction and maintenance of hospitals. It also authorizes the use of 2 per cent of the appropriations for the training of personnel necessary for such hospitals. Title can be acquired by the local County or State. The proposal expressly states that the hospitals are to be used for "diagnosis."

Kansas' Senator Arthur C. Capper is less modest in the proposals contained in his bill. Introduced, on March 25, it is the revised Epstein Bill and calls for $50,000,000 the first year and indeterminate amounts in succeeding years to induce States to establish systems of compulsory health insurance under the Social Security Act.

Senator Henry Cabot Lodge, of Massachusetts, introduced, on March 19, a bill to provide for federal health insurance program and the furnishing of "those medical services and facilities which have become standardized in their nature, but which because of high cost are not used in many cases in which their use would be unquestionably desirable."

In discussing his bill, Senator Lodge said: "I believe it is not disputed that countless instances occur every day in which x-ray examinations are desirable—nay, essential—but are not given because of the prohibitive cost. The suffering which could be prevented by prompt x-ray examination is indescribable. Needless to say, the prevention of disease automatically tends to reduce the cost of caring for the disease once it has been allowed to take hold. The use of respirators and x-rays in-

volves a technic which has become relatively standardized. In the case of the x-ray, it is not inconceivable that it would become a routine part of every physical examination were it not for the cost. In the case of persons of moderate means the cost prohibits its use. In the case of persons who can afford it, the use of x-ray is known to be so unusual that its prescription sometimes causes alarm."

A fellow Bostonian, Dr. Hugh Cabot, adds fuel to the flame by continuing his self-appointed rôle of propagandist for medical reform. He is the author of an article in the April issue of the *American Magazine*, in which he washes some of his own dirty linen and concludes that the best way to prohibit the repetitions of such ulterior practices, as he confesses, is to socialize medicine. He is apparently unaware of the national examining boards, like the American Board of Radiology, for he leaves the impression that any doctor may set himself up as a specialist without any special qualifications. He omits any mention of the fact that strenuous efforts are being made by organized medicine, without the need for governmental regimentation, to police its own members and raise the standards for specialty practice.

Says the *Journal of the Indiana Medical Association*, commenting editorially: "Dr. Cabot forgets that only a very small per cent of M.D.'s are surgeons. Most of the conditions for which patients seek relief are medical and functional—not surgical, and one of the first duties of a doctor is to relieve the patient of worry through personal contact. Dr. Cabot is not accustomed to this phase of medical practice—he is used to going to the operating room where the patient is completely ready for the surgery that he performs. He is not called upon to see the patient through the minor difficulties for which the physician collects one- and two-dollar fees. The big fees of the surgical specialist are best known to him and he has had little opportunity to know about the kind of medical practice that the average physician does—not the extremist or the ultra-conservative, but just the average physician."

MAC F. CAHAL *Executive Secretary*

RADIOLOGICAL SOCIETIES IN NORTH AMERICA

Editor's Note.—Will secretaries of societies please cooperate with the Editor by supplying him with information for this section? Please send such information to Leon J. Menville, M.D., 1201 Maison Blanche Bldg., New Orleans, La.

UNITED STATES

CALIFORNIA

California Medical Association, Section on Radiology.—Chairman, Karl M. Bonoff, M.D., 1930 Wilshire Blvd., Los Angeles; *Secretary,* Carl D. Benninghoven, M.D., 95 S. El Camino Real, San Mateo.

Los Angeles County Medical Association, Radiological Section.—President, M. L. Pindell, M.D.; *Vice-president,* Richard T. Taylor, M.D.; *Secretary,* Wilbur Bailey, M.D., 2007 Wilshire Blvd.; *Treasurer,* Henry Snure, M.D., 1414 South Hope Street; Kenneth Davis, M.D., *Member of Executive Committee.* Meets second Wednesday of each month at County Society Building.

Pacific Roentgen Club.—Chairman, Karl M. Bonoff, M.D., Los Angeles; *Members of Executive Committee,* I. S. Ingber, M.D., A. C. Siefert, M.D., D. R. MacColl, M.D.; *Secretary-Treasurer,* L. Henry Garland, M.D., 450 Sutter St., San Francisco. Executive Committee meets quarterly; Club meets annually during annual session of the California Medical Association.

San Francisco Radiological Society.—Secretary, Harold A. Hill, M.D., 450 Sutter Street. Meets monthly on third Thursday at 7:45 P.M., for the first six months at Toland Hall (Univ. of Calif. Med. School) and for the second six months at Lane Hall (Univ. of Stanford Med. School).

COLORADO

Denver Radiological Club.—President, N. B. Newcomer, M.D., 306 Republic Bldg.; *Vice-president,* Elizabeth Newcomer, M.D.; *Secretary,* Paul R. Weeks, M.D., 520 Republic Bldg.; *Treasurer,* L. G. Crosby, M.D., 366 Metropolitan Bldg. Meets third Friday of each month at homes of members.

The *Denver Radiological Club* will hold its Annual Midsummer Radiological Conference in the Rocky Mountains, with headquarters at the Hotel Shirley-Savoy, the dates of the meeting being August 8, 9, and 10.

CONNECTICUT

Connecticut State Medical Society, Section on Radiology.—Chairman, Samuel M. Atkins, M.D., 63 Central Ave., Waterbury; *Secretary-Treasurer,* Max Climan, M.D., 242 Trumbull St., Hartford. Meetings twice annually in May and September.

DELAWARE

Affiliated with Philadelphia Roentgen Ray Society.

FLORIDA

Florida Radiological Society.—President, H. B. McEuen, M.D., Jacksonville; *Vice-president,* Joseph H. Lucinian, M.D., Miami; *Secretary-Treasurer,* John N. Moore, M.D., 210 Professional Bldg., Ocala. Meetings held in November and at the annual meeting of the Medical Association of Florida in the spring.

GEORGIA

Georgia Radiological Society.—President, James J. Clark, M.D., Doctors Bldg., Atlanta; *Vice-president,* L. P. Holmes, M.D., University Hospital, Augusta; *Secretary-Treasurer,* Robert C. Pendergrass, M.D., Prather Clinic, Americus. Meetings twice annually, in November and at the annual meeting of the Medical Association of Georgia in the spring.

ILLINOIS

Chicago Roentgen Society.—President, Roe J. Maier, M.D.; *Vice-president,* Adolph Hartung, M.D.; *Secretary,* Chester J. Challenger, M.D., 3117 Logan Blvd. Meetings the second Thursday of each month from October to May, except December, at the Hotel Sherman.

Illinois Radiological Society.—President, Harry W. Ackeman, M.D., 321 W. State St., Rockford; *Vice-president,* D. R. Hanley, M.D., St. Mary's Hospital, Streator; *Secretary-Treasurer,* William DeHollander, M.D., St. John's Hospital, Springfield. Meetings quarterly by announcement.

Illinois State Medical Society, Section on Radiology.—Chairman, Warren W. Furey, M.D., 6844 Oglesby Ave., Chicago; *Secretary,* Harry W. Ackeman, M. D., 321 W. State St., Rockford.

INDIANA

The Indiana Roentgen Society.—President, Juan Rodriguez, M.D., 2902 Fairfield Ave., Fort Wayne; *President-elect,* H. H. Inlow, M.D., Shelbyville; *Vice-president,* Wemple Dodds, M.D., Crawfordsville; *Secretary-Treasurer,* Clifford C. Taylor, M.D., 23 E. Ohio St., Indianapolis. Annual meeting in May.

IOWA

The Iowa X-ray Club.—Holds luncheon and business meeting during annual session of Iowa State Medical Society.

KENTUCKY

Kentucky Radiological Society.—President, D. B. Harding, M.D., Lexington; *Vice-president,* I. T. Fugate, M.D., Louisville; *Secretary-Treasurer,*

Joseph C. Bell, M.D., 402 Heyburn Bldg., Louisville. Meeting annually in Louisville, third Sunday afternoon in April.

The Annual Meeting of the *Kentucky Radiological Society* was held at the Brown Hotel, Louisville, on Sunday, April 21, 1940. The meeting convened at 11:20 A.M. with D. B. Harding, M.D., *President*, presiding. Following the business session luncheon was served. After the luncheon Howard P. Doub, M.D., of Detroit, addressed the Society on the subject of "Childhood Tuberculosis." His presentation was illustrated by numerous lantern slides. Following Dr. Doub's address, cases illustrated by lantern slides were reported by members of the Society and were discussed by those present. The meeting adjourned at 5:30 P.M.

MAINE

See New England Roentgen Ray Society.

MARYLAND

Baltimore City Medical Society, Radiological Section.—Chairman, Harold E. Wright, M.D., 101 W. Read St.; *Secretary*, Walter L. Kilby, M.D., 101 W. Read St. Meetings are held the third Tuesday of each month.

MASSACHUSETTS

See New England Roentgen Ray Society.

MICHIGAN

Detroit X-ray and Radium Society.—President, Sam W. Donaldson, M.D., 326 N. Ingalls St., Ann Arbor; *Vice-president*, Clarence Hufford, M.D., 421 Michigan St., Toledo, Ohio; *Secretary-Treasurer*, E. R. Witwer, M.D., Harper Hospital, Detroit. Meetings first Thursday of each month from October to May, inclusive, at Wayne County Medical Society club rooms, 4421 Woodward Ave.

Michigan Association of Roentgenologists.—President, J. H. Dempster, M.D., Detroit; *Vice-president*, L. E. Holly, M.D., Muskegon; *Secretary-Treasurer*, J. E. Lofstrom, M.D., 1536 David Whitney Bldg., Detroit. Meetings quarterly by announcement.

MINNESOTA

Minnesota Radiological Society.—President, Leo G. Rigler, M.D., University Hospital, Minneapolis; *Vice-president*, Harry M. Weber, M.D., Mayo Clinic, Rochester; *Secretary*, John P. Medelman, M.D., 572 Lowry Medical Arts Bldg., St. Paul. Meetings quarterly.

MISSOURI

The Kansas City Radiological Society.—President, L. G. Allen, M.D., 907 N. 7th St., Kansas City, Kansas; *Secretary*, Ira H. Lockwood, M.D., 306 E. 12th St., Kansas City, Mo. Meetings last Thursday of each month.

The St. Louis Society of Radiologists.—President, Oscar C. Zink, M.D., St. Luke's Hospital; *Secretary*, Wilbur K. Mueller, M.D., University Club Bldg. Meets on fourth Wednesday of October, January, March, and May, at a place designated by the president.

NEBRASKA

Nebraska Radiological Society.—President, Roy W. Fouts, M.D., 1007 Medical Arts Bldg., Omaha; *Secretary*, D. Arnold Dowell, M.D., 816 Medical Arts Bldg., Omaha. Meetings third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY

(Maine, New Hampshire, Vermont, Massachusetts, and Rhode Island.) *President*, Langdon T. Thaxter, M.D., Maine General Hospital, Portland, Maine; *Secretary*, Aubrey O. Hampton, M.D., Massachusetts General Hospital, Boston. Meetings third Friday of each month from October to May, inclusive, usually at Boston Medical Library.

NEW HAMPSHIRE

See New England Roentgen Ray Society.

NEW JERSEY

Radiological Society of New Jersey.—President, P. S. Avery, M.D., Middlesex Hospital, New Brunswick; *Vice-president*, J. G. Boyes, M.D., 912 Prospect Ave., Plainfield; *Treasurer*, H. A. Vogel, M.D., 1060 E. Jersey St., Elizabeth; *Secretary*, W. James Marquis, M.D., 198 Clinton Ave., Newark; *Counsellor*, A. W. Pigott, M.D., Skillman. Meetings at Atlantic City at time of State Medical Society, and Midwinter in Newark as called by president.

NEW YORK

Associated Radiologists of New York, Inc.—President, I. J. Landsman, M.D., 910 Grand Concourse, New York City; *President-elect*, D. E. Ehrlich, M.D., 35 West 92nd St., New York City; *Vice-president*, Frederic E. Elliott, M.D., 122 76th St., Brooklyn; *Treasurer*, Solomon Fineman, M.D., 133 East 58th St., New York City; *Secretary*, William J. Francis, M.D., 210 Fifth Ave., New York City. Regular meetings the first Monday evening of the month in March, May, October, and December.

Brooklyn Roentgen Ray Society.—President, A. L. L. Bell, M.D., Long Island College Hospital, Henry, Pacific, and Amity Sts.; *Secretary-Treasurer*, L. J. Taormina, M.D., 1093 Gates Ave. Meetings first Tuesday in each month at place designated by president.

Buffalo Radiological Society.—President, Chester D. Moses, M.D., 333 Linwood Ave.; *Vice-president*, Edward C. Koenig, M.D., 100 High St.; *Secretary-Treasurer*, Joseph S. Gian-Franceschi, M.D., 610 Niagara St. Meetings second Monday evening each month, October to May, inclusive.

Central New York Roentgen Ray Society.—President, Jesse Randolph Pawling, M.D., 305 Clinton St., Watertown; *Vice-president*, Albert Lenz, M.D., 613

State St., Schenectady; *Secretary-Treasurer*, Carlton F. Potter, M.D., 425 Waverly Ave., Syracuse. Meetings are held in January, May, and October, as called by Executive Committee.

Long Island Radiological Society.—President, Samuel G. Schenck, M.D., Brooklyn; *Vice-president*, G. Henry Koiransky, M.D., Long Island City; *Secretary*, Marcus Wiener, M.D., 1430 48th St., Brooklyn; *Treasurer*, Louis Goldfarb, M.D., 608 Ocean Ave., Brooklyn. Meetings fourth Thursday evening each month at Kings County Medical Bldg.

New York Roentgen Society.—President, Harry M. Imboden, M.D., 30 W. 59th St., New York City; *Vice-president*, Henry K. Taylor, M.D., 667 Madison Ave., New York City; *Secretary*, Roy D. Duckworth, M.D., 170 Maple Ave., White Plains, N. Y.; *Treasurer*, Eric J. Ryan, M.D., St. Luke's Hospital, New York City.

Rochester Roentgen-ray Society.—Chairman, George H. S. Ramsey, M.D., 277 Alexander St.; *Secretary*, S. C. Davidson, M.D., 277 Alexander St. Meetings at convenience of committee.

NORTH CAROLINA

Radiological Society of North Carolina.—President, Robert P. Noble, M.D., 127 W. Hargett St., Raleigh; *Vice-president*, A. L. Daughtridge, M.D., 144 Coast Line St., Rocky Mount; *Secretary-Treasurer*, Major I. Fleming, M.D., 404 Falls Road, Rocky Mount. Meetings with State meeting in May, and meeting in October.

OHIO

Cleveland Radiological Society.—President, L. A. Pomeroy, M.D., Hanna Bldg., Cleveland; *Vice-president*, P. C. Langan, M.D., 215 Wellsley Ave., Akron; *Secretary-Treasurer*, H. A. Mahrer, M.D., 10515 Carnegie Ave., Cleveland. Meetings at 6:30 P.M. at the Mid-day Club, in the Union Commerce Bldg., on fourth Monday of each month from October to April, inclusive.

A notice of the joint meeting with the Pittsburgh Roentgen Society will be found under the latter Society, on this page.

Radiological Society of the Academy of Medicine (Cincinnati Roentgenologists).—*President*, Archie Fine, M.D., 707 Race St., Cincinnati; *Secretary-Treasurer*, Justin E. McCarthy, M.D., 707 Race St., Cincinnati, Ohio. Meetings held third Tuesday of each month.

PENNSYLVANIA

Pennsylvania Radiological Society.—President, Louis A. Milkman, M.D., Medical Arts Bldg., Scranton; *First Vice-president*, James E. Ginter, M.D., Dubois; *Second Vice-president*, Alexander Stewart, M.D., Shippensburg; *Secretary-Treasurer*, L. E. Wurster, M.D., 416 Pine St., Williamsport; *President-elect*, Harvey N. Mawhinney, M.D., 6546 Darlington Road, Pittsburgh; *Editor*, William E. Reiley, M.D., Clearfield; *Assistant Editor*, Sydney J. Hawley, M.D., Danville.

The Philadelphia Roentgen Ray Society.—President, Joseph E. Roberts, Jr., M.D., 403 Cooper St., Camden, N. J.; *Vice-president*, Jacob H. Vastine, M.D., Medical Arts Bldg., Philadelphia; *Secretary*, Barton R. Young, M.D., Temple University Hospital, Philadelphia; *Treasurer*, Fay K. Alexander, M.D., Chestnut Hill Hospital, Philadelphia. Meetings held first Thursday of each month at 8:15 P.M., from October to May, in Thomson Hall, College of Physicians, 21 S. 22nd St., Philadelphia.

The Pittsburgh Roentgen Society.—President, Zoe A. Johnston, M.D., 601 Jenkins Arcade; *Vice-president*, Prentiss A. Brown, M.D., and *Secretary-Treasurer*, Harold W. Jacox, M.D., 4800 Friendship Ave. Meetings held second Wednesday of each month at 4:30 P.M., from October to June at various hospitals designated by program committee.

On Monday, April 29, the members of the Cleveland Radiological Society were the guests of the *Pittsburgh Roentgen Society* at a meeting and dinner in the Hotel William Penn in Pittsburgh. Fifty-six members and guests attended and an excellent scientific program was presented.

Dr. Lloyd Craver, of the *Memorial Hospital*, New York City, spoke on the subject of Bronchiogenic Carcinoma. Dr. Charles Higley and Dr. Harry Hauser, of the Cleveland City Hospital, spoke on the Diagnosis and Treatment of Intrathoracic Hodgkin's Disease, respectively. Dr. Paul Bovard, of Tarentum, Pa., presented Diagnostic Problems Associated with Silicosis.

It is hoped that a combined meeting of the two societies will become an annual affair.

RHODE ISLAND

See New England Roentgen Ray Society.

SOUTH CAROLINA

South Carolina X-ray Society.—President, T. A. Pitts, M.D., Columbia; *Secretary-Treasurer*, Malcolm Mosteller, M.D., Columbia Hospital, Columbia. Meetings in Charleston on first Thursday in November, also at time and place of South Carolina State Medical Association.

SOUTH DAKOTA

Meets with Minnesota Radiological Society.

TENNESSEE

Memphis Roentgen Club.—Chairmanship rotates monthly in alphabetical order. Meetings second Tuesday of each month at University Center.

Tennessee Radiological Society.—President, Steve W. Coley, M.D., Methodist Hospital, Memphis; *Vice-president*, Eugene Abercrombie, M.D., 305 Medical

Arts Bldg., Knoxville; *Secretary-Treasurer*, Franklin B. Bogart, M.D., 311 Medical Bldg., Chattanooga. Meeting annually with State Medical Society in April.

TEXAS

Texas Radiological Society.—President, C. F. Crain, M.D., Corpus Christi; *President-elect*, M. H. Glover, M.D., Wichita Falls; *First Vice-president*, G. D. Carlson, M.D., Dallas; *Second Vice-president*, P. E. Wigby, M.D., Dallas; *Secretary-Treasurer*, L. W. Baird, M.D., Scott and White Hospital, Temple. Meets annually. The next annual meeting is to be Jan. 18, 1941, in Sherman.

VERMONT

See New England Roentgen Ray Society.

VIRGINIA

Radiological Society of Virginia.—President, Fred M. Hodges, M.D., 100 W. Franklin St., Richmond; *Vice-president*, L. F. Magruder, M.D., Raleigh and College Aves., Norfolk; *Secretary*, V. W. Archer, M.D., University of Virginia Hospital, Charlottesville.

WASHINGTON

Washington State Radiological Society.—President, H. E. Nichols, M.D., Stimson Bldg., Seattle; *Vice-president*, George Cornett, M.D., Yakima; *Secretary-Treasurer*, Kenneth J. Holtz, M.D., American Bank Bldg., Seattle. Meetings fourth Monday of each month at College Club, Seattle.

WISCONSIN

Milwaukee Roentgen Ray Society.—President, H. W. Hefke, M.D.; *Vice-president*, Frederick C. Christensen, M.D.; *Secretary-Treasurer*, Irving I. Cowan, M.D., Mount Sinai Hospital, Milwaukee. Meets monthly on first Friday at the University Club.

Radiological Section of the Wisconsin State Medical Society.—Secretary, Russel F. Wilson, M.D., Beloit Municipal Hospital, Beloit. Two-day annual meeting in May and one day in connection with annual meeting of State Medical Society, in September.

University of Wisconsin Radiological Conference.—Secretary, E. A. Pohle, M.D., 1300 University Ave., Madison, Wis. Meets every Thursday from 4 to 5 P.M., Room 301, Service Memorial Institute.

CANADA

Section on Radiology, Canadian Medical Association.—Chairman, Gordon Richards, M.D., Medical Arts Bldg., Toronto; *Secretary*, W. J. Cryderman, M.D., Medical Arts Bldg., Toronto.

Section on Radiology, Ontario Medical Association.—Chairman, E. H. Shannon, M.D., St. Michael's Hospital, Toronto; *Secretary*, W. J. Cryderman, M.D., 474 Glenlake Avenue, Toronto.

Canadian Association of Radiologists.—President, J. E. Gendreau, M.D., Montreal; *Vice-president*, W. H. McGuffin, M.D., Calgary; *Honorary Secretary-Treasurer*, W. L. Ritchie, M.D., Montreal; *Chairman of Interrelations Committee*, G. E. Richards, M.D., Toronto.

EDITORIAL

LEON J. MENVILLE, M.D., *Editor* HOWARD P. DOUB, M.D., *Associate Editor*

THE RADIOLOGIST'S PLACE IN THE DIAGNOSIS OF LUNG PATHOLOGY

A few years ago, when the radiologist was requested to make an x-ray examination of the chest and express an opinion, too often his findings were either accepted by the surgeon, internist, or general practitioner, as the final word and allowed to override conflicting clinical evidence, or they were minimized and the clinical evidence overemphasized. Twenty-five years ago, there were many good clinicians who sincerely believed that a careful physical examination by a competent chest man would afford an accurate diagnosis in most cases of early tuberculosis. They certainly believed that physical signs were far more important than the x-ray findings. As the fundamental knowledge on which an accurate interpretation of chest pathology as revealed in the roentgenogram became more widely known, the well trained clinician quickly recognized the important place which an x-ray examination of the chest occupied. Conversely, the good radiologist soon appreciated that, even if a great deal of early pathology in the lungs, which was overlooked by the physical examination, was detected by the roentgen examination, the x-ray examination in no way replaced the physical examination, and clinical history but only supplemented them.

As the years have passed, the proper place of the x-ray in the examination of the chest has been recognized by the great majority of both clinicians and radiologists. Unfortunately, there are still some clinicians and some men who are doing radiology, who do not appreciate what the proper place of the x-ray examination is in the determination and evaluation of chest pathology. About ten years ago, a patient was referred to the writer for an x-ray examination of the chest by a general practitioner. The findings were such as were at that time generally ascribed to healed miliary tuberculosis, although now we recognize the fact that they are likely produced by other processes. In the description of the film, the statement was clearly made that there was no soft infiltration, which was suggestive of active tuberculosis. In the space at the bottom of the report sheet reserved for the roentgen diagnosis, this statement was made, "healed miliary tuberculosis." Unfortunately, only the written report was rendered, and the case was not discussed with the clinician. A few weeks later, it was learned that the doctor had hurriedly glanced at the diagnosis and, overlooking the word "healed," had sent the patient to a tuberculosis sanatorium with a diagnosis of tuberculosis based on the x-ray findings.

This incident not only illustrates the fact that clinical diagnoses should be arrived at after consultation between the clinician and the radiologist, but that the same kind of report is not suitable when rendered to different types of referring physicians. In a large closed hospital or clinic in which there exist close understanding and co-operation between the different departments, a detailed description of the roentgen findings may not be necessary in every case, and only the doubtful cases need be carefully discussed in conference. In other institutions, where the size of the staff permits, it might well be argued that an accurate description should be rendered by the radiologist and that only in conference should any diagnosis be made. In the average community the problem of the radiologist may be quite different. He will undoubtedly have many referring physicians with whom he has a perfect understanding who will evaluate the x-ray report at its true worth and who will gladly consult with the radiologist in all doubtful cases. In spite of all the educational work which the competent radiologist can diplomatically do, there will be a considerable number of referring physicians who, because of indifference or haste, will depend on the radiologist's report almost exclusively to arrive at a clinical diagnosis. There will also be a few men who will fail to realize the necessity

for an x-ray examination and will not have it done unless the patient insists. Such a doctor will be apt to minimize the importance of the x-ray findings. Fortunately, such doctors are not so frequently encountered as they were a few years ago.

Another thing that must be taken into account in evaluating the proper place of the roentgen examination in the diagnosis of chest pathology is the fact that with the great increase in the number of cases coming for roentgen examination, and the widespread propaganda for early diagnosis, many early lesions are being encountered that were formerly not seen. The experienced radiologist encounters an increasing number of cases in which it is not only necessary to correlate the roentgen and clinical findings, but in which a diagnosis is possible only after repeated roentgen examinations at fairly frequent intervals and careful clinical checks.

With the recent widespread distribution of x-ray equipment and the interpretation of chest films by the comparatively inexperienced, shadows on the film that are of no particular significance are frequently interpreted as indicating the presence of grave pathology. This mistake occurs more commonly than for a serious lesion to be overlooked.

The good radiologist experiences no chagrin in frankly stating in a given case that he does not know how to interpret the shadows seen because they are unusual and represent a type of change with which he is not familiar. For instance, a radiologist might be quite familiar with changes produced by tuberculosis and pneumonia and the usual neoplastic growths that occur in the lungs, but might not have had the opportunity of seeing a group of unusual lung tumors and might not be located in a community where silicosis is commonly encountered. Consequently, he would feel no hesitancy in seeking aid when a film presented itself with changes which he did not understand and which he suspected of representing some unusual condition.

We might conclude that the radiologist's place in the diagnosis of lung pathology is that of an important consultant whose findings must be correlated with those of the other medical men. His exact position will vary considerably in different communities and even in the same community, as he deals with different types of referring physicians. He should state his findings and opinions boldly, but he should have no false pride and should frankly state his inability to understand the changes seen in any given case and should seek aid when a group of changes are encountered with which he is not familiar. The radiologists, as well as other medical men, are constantly seeing more early pathology because of the widespread propaganda for routine examinations and because of the more general use of roentgen methods of examination. This fact should stimulate all of us to seek better criteria on which to base an early diagnosis of the unusual lesion which we encounter.

FRANKLIN B. BOGART, M.D.
Chattanooga, Tenn.

COMMUNICATION

TESTIMONIAL DINNER TO DR. I. SETH HIRSCH

On April 17, 1940, in the Hotel Biltmore, New York City, was held a testimonial dinner by the friends and associates of Dr. I. Seth Hirsch. The speakers were Currier McEwen, M.D., Dean of the College of Medicine, New York University; Miles Sturtevant, M.D., of New York City; Harry E. Isaacs, M.D., of New York City; Charles Gottlieb, M.D., of New York City; Henry M. Silver, M.D., of New York City; Arial W. George, M.D., of Boston; Bernard H. Nichols, M.D., of Cleveland, Ohio, President of the Radiological Society of North America; Arthur M. Wright, M.D., of New York City, and A. A. Brill, M.D., of New York City. The Dinner Committee consisted of Charles Gottlieb, M.D., Harry E. Isaacs, M.D., Maxwell H. Poppel, M.D., and Henry K. Taylor, M.D. The Radiological Society of North America was represented by Bernard H. Nichols, M.D., President, and Donald S. Childs, M.D., of Syracuse, N.Y., Secretary-Treasurer. Dr. Hirsch is likewise a member of this Society, as were many of the other guests.

Considerable additional interest was contributed to the occasion by the production of a scene from a play, "The Unlistening Street," written by Dr. Hirsch and Evans Nash. Professor Otto Glasser, Ph.D., of Cleveland, Ohio, spoke wittily on "Roentgen's Discovery in Contemporary Humor." Music was furnished by the Moss String Quartet.

IN MEMORIAM: THOMAS ALLEN GROOVER, M.D.

Thomas Allen Groover was born at Pidcock, in southern Georgia, on May 1, 1877, a son of Thomas Alfred and Sarah Jane (Joiner) Groover. His father, who was a farmer, was a Confederate soldier during the Civil War and had lost an arm in action. Dr. Groover received his early education in the public schools of Brooks County, Georgia. Because of financial considerations it did not seem possible for him to continue his education, but through the good offices of the Hon. Hoke Smith, of Georgia, who was Secretary of the Interior in President Cleveland's Cabinet, he was appointed as assistant messenger in the Department of the Interior. He then came to Washington, in 1893, and resided there all the remainder of his life. In 1894, he entered the Medical Department of Columbian University, which later became The George Washington University, and graduated therefrom in 1898. In 1926, his alma mater conferred upon him the degree of Doctor of Science. After internship at the Garfield Memorial Hospital he was appointed physician with the Isthmian Canal Commission and in that capacity spent the next year in Nicaragua.

Dr. Groover returned to Washington, in 1900, and entered the general practice of medicine. At the same time he began x-ray work at the Central Dispensary and Emergency Hospital. Although he did not completely specialize in radiology until 1912, he was actively engaged in that field from his entry into it, in 1900, up to within three months of his death which occurred on April 20, 1940. It was in those early days from 1900 to about 1904 or 1905 that he suffered the injuries to his hands which resulted in amputation through the left forearm, in 1926, and to final involvement of the right axilla and lung which cost him his life.

Dr. Groover was closely associated with the scientific progress and the organizational development of radiology in this country during the entire first forty years of the present century. He made numerous contributions to the literature and was always interested and quietly helpful in everything that affected the welfare of the specialty that he loved. He was insistent, however, that the radiologist is primarily a physician, and as such he always established a personal relationship with every patient who came under his care. His membership in medical organizations included the following: Medical Society of the District of Columbia, of which he was President in 1925; Fellow of the American Medical Association; Fellow of the American College of Physicians; member of the Southern Medical Association, of which he was Vice-president in 1924; member of the American Roentgen Ray Society and its President in 1925; member of the Radiological Society of North America; Fellow of the American College of Radiology, in which he served both as President and as Chancellor.

Dr. Groover's work as a practising radiologist was characterized by the utmost painstaking care and attention to detail. He not only required the best possible technical work but spared no time nor personal effort to arrive at a correct interpretation and final diagnosis. He had developed in himself to a very high degree those qualities which are indispensable in the good physician—accuracy in observation and a keen sense of relative values. He was a diagnostician of unusual ability. In manner Dr. Groover was quiet and reserved. Being of a very studious nature, he made it a rule to spend at least one hour a day in the reading of medical literature. He had great qualities both of mind and heart which were unusual and outstanding. His ability as an organizer and executive was well known. His careful, methodical attention to all the details of bookkeeping, records, and management laid the foundation for his professional work. He always looked upon business arrangements, however, as a means to more important ends. He was first and last a physician. Even radiology, in every branch of which he was proficient, he insisted must always be looked upon as an integral part of general medical practice. His outstanding mental characteristics were patience and a great capacity for methodical, painstaking care in every diagnostic and therapeutic procedure. His dominant moral characteristic was



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The late THOMAS A. GROOVER, M.D.

a keen sense of justice, right, and fair dealing. He had an unfailing patience in compromising differences of opinion among his associates, a sane judgment in arriving at important decisions, and above all, a kindly, tolerant charity toward all with whom he could not agree. He will be greatly missed by his close associates who were accustomed to call upon him almost daily for his helpful counsel, and his loss will be keenly felt in many organizations in which his advice was highly valued. His death adds another martyr to the long roll of those who have sacrificed their lives in the interests of science and humanity.

ARTHUR C. CHRISTIE, M.D.
Washington, D. C.

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JOHN M. MILES, M.D., of Lafayette, La.
R. R. NEWELL, M.D., of San Francisco, Calif.
LESTER W. PAUL, M.D., of Madison, Wisc.
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LARYNX

Laryngocele. John Blewett. British Jour. Radiol., 12, 163–167, March, 1939.

At the anterior end of the sinus of the larynx is the small saccule. The thyro-hyoid membrane over the saccule is strengthened at the middle and at the lateral edges but between these places it is weak. The superior laryngeal vessels enter here, which further weakens this spot. When the saccule becomes distended with air, there is little to prevent the formation of a laryngocele. The condition is usually found in those who use the voice excessively or in glass blowers and wind-instrument players. The cyst can remain in the larynx or it may herniate into the neck. Sometimes it appears only on forced phonation and then disappears, but in some cases it may remain and obstruct respiration. Roentgenographic examination has been seldom used in the diagnosis. The appearance of the air-filled tumor on the film is striking, so the diagnosis can be readily made by x-ray examination. An illustrative case is presented.

SYDNEY J. HAWLEY, M.D.

X-ray Examination of Larynx and Pharynx. R. P. O'Bannon. Texas St. Jour. Med., 34, 849–851, April, 1939.

The radiographic anatomy and physiology of the soft tissues of the neck are reviewed and the value of radiographic study in pathologic conditions pointed out.

A lateral view of the neck is made without preliminary preparation by the use of a soft-tissue technic. Inflammatory or neoplastic diseases of the pharynx or larynx show an alteration in the shape and size of the soft-tissue structures which stand out in contrast to the air in this region.

The information obtained is often greater and more specific than that obtained on clinical inspection. It is also an excellent record of the progress or healing of the diseased condition.

JOHN M. MILES, M.D.

Roentgen Consideration of Lesions in and about the Larynx: Diagnostic Aspects. Adolph Hartung. Illinois Med. Jour., 76, 125–127, August, 1939.

According to the author, far too little use is made of the x-ray and fluoroscope when considering lesions in and about the larynx—a region which is especially favorable for roentgen examination because of the air passages. Fluoroscopic examination with sagittal and lateral views, with films offering a maximum of soft-tissue contrast, is advised. In some cases opaque media are advantageous. Intralaryngeal films as used by Waldapfel and special procedures such as drawing the larynx forward by means of a curved sound, or overdistending the air passages by the Valsalva method as suggested by Jönnson, are also to be considered. The profession must become more appreciative of the value of these methods, for in some cases they may be the only practicable means of obtaining the necessary information as to the nature, exact location, and extent of a lesion. They should especially be considered in the presence of such complaints as hoarseness, aphonia, difficulty of speech, stridor, recurrent cyanosis, strangling attacks, lump in the throat, dysphagia or pain on swallowing, pain or swelling in anterior part of neck, and suspicion of foreign bodies. The non-radiopaque foreign bodies may cause recognizable soft-tissue changes to reveal their location and a follow-up examination is advisable to rule out the possibility of stricture. Trauma is also an indication, as displacements of parts of the larynx or fractures of the cartilaginous portions may be found, especially if ossification is present.

Inflammatory conditions of the neck, especially retropharyngeal abscess, are to be noted. Conditions which produce narrowing of the lumen of the air passages by external compression, such as post-inflammatory stenosis, thyroid enlargements, tumors, and other swellings in the neck region, may be located.

Tuberculosis, syphilis, benign lesions, tumors of the base of the tongue, lesions of the cervical portion of the esophagus, and neoplasms may be discovered by roentgenographic examination. This is not only an aid in diagnosis, but may suggest the preferable means of treatment, or the site for radon implantation, or topical application of radium. Furthermore, tracheotomy procedures and the proper fitting of tracheotomy tubes may be aided by roentgenographs of the neck. The author pleads for a greater appreciation of the value of these methods, a greater dissemination of the knowledge of such methods, and a more frequent use of them. He reminds us that a chest examination is often incomplete without a detailed examination of the neck, since that is where the trouble may be. Attention is called to the necessity of correlating the clinical findings with the roentgen findings and with those of the laryngologist.

WILLIS A. WARD, M.D.

Roentgen Consideration of Lesions in and about the Larynx. T. J. Wachowski. Illinois Med. Jour., 76, 128–130, August, 1939.

The author divides the lesions of the larynx which warrant therapeutic irradiation into those of inflammatory and neoplastic origin. For the inflammatory ones, he advises judicious small dosage irradiation, especially when there is perilaryngeal involvement of the phlegmonous type.

For lesions of neoplastic origin, the author states that the benign ones are ordinarily handled by surgery, but in the field of malignant neoplasms there is a great need of advancement of therapy. Irradiation may be applied internally or externally, but the former method has met with little success. The latter method is the one with which we are more concerned and the application may be by either high voltage roentgen rays or the radium bomb. No significant difference in results has been reported thus far.

Irradiation is accepted as the method of choice in all inoperable cases. The accepted rationale of the protracted intensive method is the differential action of

gamma rays on the more undifferentiated cells of the body. It is conceded that the curability of carcinoma of the larynx by irradiation is greatest in the intrinsic lesion and decreases as the growth is removed farther from the vocal cords. Mention is made of a group of radioresistant lesions.

For the technic one must consider the size of the lesion, the age and condition of the patient, and the presence or absence of metastases and infection. If infection is present, it is advisable to precede the main course of therapy by a short period (13 to 26 days) of treatment given in small dosages (5 to 50 r) through large portals. This is intended to control or eliminate the infection and to modify favorably the vasculoconnective tissue. The cellulocidal therapy is best delivered through the smallest portals possible so as to be more likely to irradiate the lesion sufficiently before endangering the general condition of the patient. Round portals are considered better than square ones.

The indications are not clear-cut for either surgery or irradiation, but mention is made of Coutard's classification of laryngeal carcinomas into lesions composed of undifferentiated cells and those composed of differentiated cells. Surgery is recommended for the differentiated type since cure by irradiation is considered almost impossible. But in the undifferentiated group, irradiation therapy is the method of choice in all cases, since it is never biologically operable because of early and widespread metastasis. If surgery should be insisted upon, pre-operative irradiation is advised.

The main course of irradiation should be planned so as to deliver the calculated dose in about 25 or 30 days. Protraction of therapy over more than 60 to 90 days is to be guarded against because this introduces the risk of "radio-vaccination," a condition of increased radioresistance of the tumor cells. Most of the successfully treated cases, in the author's experience, using portals of average size (50 sq. cm. ±), fell within a certain dosage zone, usually 4,000 r × 2 (measured in air).

WILLIS A. WARD, M.D.

The Treatment of Cancer of the Larynx, with Comparison of Results Obtained by Surgery and Radiation Therapy. M. F. Arbuckle. Southern Med. Jour., 32, 1008–1012, October, 1939.

Deaths from carcinoma of the larynx constitute 1.8 per cent of all cancer deaths in the United States. The treatment to be employed varies with the location, extent, and degree of malignancy. Authorities differ as to the form of therapy most advisable.

The site of origin of laryngeal carcinoma most often is the true vocal cord and, since metastases do not occur while the tumor is confined to the cord, surgical removal of cordal cancer results in a lasting cure in 80 per cent of cases, with little inconvenience, low mortality, and no loss of function. Irradiation is less satisfactory in this type of cancer than is surgery.

In other types of laryngeal cancer with lymphatic involvement the results of radiation therapy alone are better than those obtained by surgical treatment. It is a mistaken idea, however, that suffering, mutilation, and danger to life are greater from surgery than from irradiation, particularly when the radiation is administered to the syphilitic, the aged, or the infirm.

The results of 34 cases of carcinoma of the larynx are tabulated, and a typical case of intrinsic carcinoma and one of an extrinsic carcinoma of the larynx are reported in detail.

JOHN M. MILES, M.D.

LEUKEMIA

Roentgen Therapy of Leukemia with Variable Filters. A. Possati. Strahlentherapie, 65, 54, 1939.

In 1925, Ghilarducci recommended a technic of roentgen irradiation with variation of filters during a course of treatments. The author has used a modification of this plan in cases of leukemia. He employed from 180 to 200 kv., 2 ma. and doses of one-thirtieth to one-tenth S.E.D. The fractional doses are given with the filter with 1 mm. Al, with 3 mm. Al, with 5 mm. Al, and then with 0.5 mm. Zn + 3 mm. Al, exposing two large fields, the liver and spleen. Often Possati saw striking response even in late stages which had become fairly radioresistant, and he proposes, therefore, to give this method a trial in the treatment of deep-seated malignant neoplasms.

ERNST A. POHLE, M.D., Ph.D.

The Symptomatology of Lymphoma: Its Endless Variety. J. H. Means. Jour. Am. Med. Assn., 113, 646–649, Aug. 19, 1939.

Lymphoma is a disease both ubiquitous and pleomorphic, presenting itself in ever-changing symptomatology. It has a great capacity for producing bizarre clinical pictures and a propensity for being heralded by signs and symptoms diagnostically misleading.

The disease is generally considered to be neoplastic—a neoplasm taking origin from a cell of the lymphocytic series anywhere between the primordial, or stem cell, and the mature cellular elements. Pathologists recognize several types described by such terms as lymphoblastoma, reticulum-cell sarcoma, lymphosarcoma, Hodgkin's disease and giant follicular lymphoma. Whether these types represent separate entities or merely varieties of a single entity is undecided. That in certain cases the pathologic picture may pass from one type to another suggests that the disease is fundamentally unitary.

All types are, relatively speaking, radiosensitive and yield to some extent for a time to rather low dosage of radiation.

It has been shown that rate of progress of the disease bears some relation to the anatomic picture. Life expectancy is longer when the anatomic picture called "giant follicular lymphoma" is present than in any of the others. That type is seemingly the least malignant.

Although fundamentally neoplastic, the disease lymphoma resembles more closely in its clinical course and behavior certain chronic infections than it does other malignant tumors. While it may start locally in

one particular lymph node or other tissue where cells of the lymphatic series are present, its inherent tendency is to become systemic or even constitutional. In most cases, at some stage of the disease, the course is febrile.

The symptoms of the disease may be the result of local interference with the function of involved organs or of pressure on neighboring ones, or of the general effects of the systemic extension of the lymphomatous process, such as fever, fatigue, or itching. Since almost any part, or parts, of the body may be involved, the symptoms and signs which may be produced are nearly endless.

The absolute diagnosis of lymphoma can be made only by biopsy. A negative biopsy does not exclude the existence of lymphoma in the patient. The x-ray appearance of intrathoracic or gastro-intestinal lesions sometimes gives almost uncontrovertible evidence, and the rapid melting of a lesion under x-radiation leaves only a very small, if any, doubt as to its lymphomatous nature. Subsidence of fever under such treatment has a similar significance.

CHARLES G. SUTHERLAND, M.D.

THE LUNGS

Hydatid Cysts in Both Lungs. H. Kenrick Christie. Australian and New Zealand Jour. Surg., 8, 373–378, April, 1939.

In reviewing the records of the Australasian Hydatid Registry, of 1,350 cases, the author collected 250 cases of lung cysts, of which 16 were bilateral. He discusses in detail the symptomatology, surgical treatment, and prognosis of this rare condition and presents numerous excellent radiographs to illustrate his case reports. The similarity between hydatid cysts of the lung and large metastatic tumors (such as metastatic hypernephroid carcinoma) is striking.

SIMON POLLACK, M.D.

Transitory Lung Infiltrations Associated with Eosinophilia (Löffler's Syndrome): Report of a Case. D. C. Wharton Smith and Alexander J. Alexander. Southern Med. Jour., 32, 267–272, March, 1939.

An atypical and controversial case is reported, characterized by diffuse transitory areas of density in the lungs (lasting only 14 days), eosinophilia, acute illness, and death, in two months, of a seven-year-old white girl. A complete autopsy report is included. The diagnosis is unsettled because of the complicating features. Löffler's syndrome, septicemia, and leukemia are considered.

JOHN M. MILES, M.D.

Bronchographic Study of Apparently Healed Lung Abscesses. R. Maxwell Franklin. Am Jour. Med. Sci., 198, 95–98, July, 1939.

Bronchographic study after the disappearance of the symptoms and the roentgenographic shadows of acute suppurative bronchopulmonary disease often reveals residual pathologic defects in the lungs and bronchi that are otherwise unrecognizable.

Bronchography should be used routinely to determine the status of the bronchi and lungs after the symptoms and roentgenographic shadows of the acute suppurative process have disappeared.

The clinical concept of healing of acute suppurative bronchopulmonary processes should be broadened to include the residual pathologic changes in the lungs and bronchi.

BENJAMIN COPLEMAN, M.D.

METASTASIS

Skeletal Metastasis in Cancer. Charles F. Geschickter and I. H. Maseritz. Jour. Bone and Joint Surg., 11, 314–322, April, 1939.

These authors have reviewed 5,739 cases of carcinoma arising from various organs and have recorded the incidence and location of metastasis to bone as found on x-ray examination. Of this number, 356 showed metastasis to bone, and in 60 cases (16.8 per cent) the primary site of the growth was not located. Pathologic fractures occurred in 19 instances (31.6 per cent); solitary lesions occurred in 34 (56.6 per cent).

Metastatic hypernephroma occurred in 41.5 per cent; bone metastasis from breast cancer, 5.6 per cent; in cancer of the prostate, 13 per cent; urinary bladder cancer, 47 per cent; one in 80 cases of carcinoma of the esophagus showed bone metastasis; seven of 750 cases of carcinoma of the stomach; three of 620 cases of carcinoma of the large intestine and rectum; one in 46 cases of carcinoma of the liver; six of 76 cases of carcinoma of the thyroid; five in 86 cases of carcinoma of the uterus.

The spine and pelvis are the most frequent sites of bone metastasis, but other bones may also be involved. Irradiation of metastatic lesions relieves pain and prolongs life.

J. B. MCANENY, M.D.

Metastasis to Bone from Carcinoma of the Gastro-intestinal Tract. Ralph K. Ghormley and Jorge E. Valls. Jour. Bone and Joint Surg., 21, 74–78, January, 1939.

Review of the pertinent literature reveals that autopsy studies place the figure of bone metastasis from gastro-intestinal carcinoma too high, because of the terminal state of the disease.

Most metastases are seen in the spine, pelvis, and ribs, the long bones being but rarely involved. The incidence of metastasis to bone is found to be between 0.2 and 0.5 per cent of cases.

JOHN B. MCANENY, M.D.

PELVIC MEASUREMENTS

Stereoscopic Measurements of Distances in the Female Pelvis and on the Fetal Skull. Benedict Westergaard. Acta Radiol., 20, 33–39, February, 1939.

The author reports his experience and findings in stereoscopic measurements of the maternal pelvis and the fetal head. From frontal roentgenographs, the su-

perior transverse diameter and the distance between the ischial spines were determined, occasionally also a minimum value of the conjugata vera. However, the conjugata vera was, as a rule, determined from profile roentgenographs. As far as the fetal head is concerned, its biparietal and bitemporal diameters can be ascertained only when they lie at right-angles to the central rays from the two foci. The stereoscopic measurements of the conjugata vera were less accurate than those of the fetal skull. While in the latter case the error amounted to less than 1 mm., the difference between roentgen measurements of the conjugata vera and check measurements taken by the direct method during laparotomy was higher, though never exceeding 3.4 mm.

ERNST A. SCHMIDT, M.D.

Pelviradiography and Clinical Pelvimetry: Comparative Values in the Prognosis of the Outcome of Labor. Emanuel M. Rappaport and Samuel J. Scadron. Jour. Am. Med. Assn., 112, 2492–2497, June 17, 1939.

X-ray examinations were originally employed for the purpose of attesting fetal presence, multiple fetus, and fetal deformities. During the last fifteen years several methods of x-ray pelvimetry have been introduced.

This study covers a series of 350 patients, the x-ray examination being made ten days prior to the calculated date of labor. In instances of miscalculation, when labor was delayed several weeks, x-ray examination was repeated at term.

In the classification of the various types of pelves, such factors as size of inlet, relationship between head and inlet, sacrosciatic notch, inclination of sacrum and lateral bone, sacral promontory, subpubic angle, shape of arch, length of rami and side walls of the pelvis were taken into consideration. The classification adopted was that of Caldwell and Moloy. In the series studied, the percentages of the various major pelvic types closely approximated those of other observers, with the exception of the platypelloid type.

The gynecoid pelvis is the type most frequently encountered in any series; it occurred in 158 of these 350 cases. The anthrapoid type was next in frequency, with 52 cases. Forty-four women in this series showed the true android type of pelvis, and 23 the mixed android type (*i.e.*, with android posterior segments and gynecoid forepelvis). There were 12 cases of true platypelloid pelvis and nine cases of mixed type. The authors explained the more frequent occurrence of this type in their series by the fact that their patients were entirely those of the Jewish race, wherein females of the hyposthenic and pyknic type are frequently encountered.

The types were charted to indicate the spontaneous deliveries, the elective forceps deliveries, and the instrumental deliveries in each.

The authors felt that clinical experience without the use of pelviradiography has the advantage over the use of x-ray interpretation alone, and that it was of paramount importance that the obstetrician develop himself clinically rather than to rely solely on x-ray methods. The greatest acumen in determining the relative size of the passage and passenger and in selecting the appropriate operative procedure is best obtained when the experienced obstetrician combines his clinical knowledge with pelviradiography.

CHARLES G. SUTHERLAND, M.D.

PEPTIC ULCER

A Frequent Non-ulcerous Cause of the Peptic Ulcer Syndrome. Lester M. Morrison, William A. Swalm, and Chevalier L. Jackson. Pennsylvania Med. Jour., 43, 243–247, December, 1939.

Gastritis may be the only pathology found in 30 per cent of patients exhibiting the textbook picture of peptic ulcer. The symptoms of gastritis are so variable that they may range from complete absence of symptoms to the perfect mimicking of peptic ulcer with its food or alkali relief, and hematemesis. Deformity of the duodenal cap has been seen on x-ray examination, which, at operation, has proven to be due entirely to gastritis.

JOSEPH T. DANZER, M.D.

Ulcerative Gastritis and Residual Lesions. H. E. Robertson. Jour. Am. Med. Assn., 112, 22–27, Jan. 7, 1939.

The stomach represents a very complex physiologic mechanism affected almost as frequently and as severely by extrinsic as by intrinsic factors. When an extrinsic influence, such as a lesion in the brain or a severe anemia, upsets this mechanism the resulting disturbances may in turn bring about local changes. If one injects the arterial trunk supplying the gastric mucosa with any substance which permits the capillaries to be visualized, they will appear in the mucous membrane as an extremely thick network so numerous that, by dilating them with India ink, one will see that the surface resembles a solid mass of this black substance. Whenever, for any reason, an abnormal congestion of these capillaries occurs, one receives the same impression of a tremendously rich capillary blood supply to the entire mucosa. In ordinary microscopic preparations, even of freshly preserved normal stomachs, these capillaries are but inadequately seen, but their importance in the interpretation of many lesions in the stomach cannot be overestimated.

Apparently whenever congestion of the capillary network occurs, even with the normal physiologic stimuli of digestion, a slight over-distention in any local area may bring about a rupture of one or more capillary loops. As soon as the hemorrhage occurs, necrosis of the involved part is an inevitable result. This necrosis is promptly digested and leaves an ulcer, which ordinarily is promptly repaired with complete restoration of the integrity of the mucosa. In most instances the entire process is without a single discoverable clinical sign or symptom.

While many small hemorrhages and their resulting ulcers may be followed by complete restitution, often there remain more or less definite gross or microscopic evidences of the fact that, after one or more local areas

in the stomach have previously suffered from ulcers, imperfect instead of perfect healing has resulted.

When hemorrhage in the mucosa produces necrosis, the production of mucin in that area ceases and the necrotic area is promptly digested until the living glands are exposed and fresh mucous reserves can once more inhibit the action of the digestive ferments. The lack of sufficient mucin as to either quality or quantity may thus account for the failure of proper or prompt healing of certain ulcers. The possibility that some nervous mechanism, definitely affected by cerebral conditions, may play a rôle in the elaboration of this substance has to be seriously considered.

When ulcers are deeper and more severe, after healing, a stellate scar may persist as a residual gross lesion. Atrophic processes or hyperplastic changes may result. Under certain circumstances hyperplastic changes in the mucous glandular cells may proceed to the stage of autonomous newgrowth. Carcinomas probably most commonly have their origin in atrophies, disorganizations and reparative hyperplasias which represent the residuals of many previous ulcers.

The clinician should not use the almost meaningless term "chronic gastritis." Instead, he should employ names which have a definite significance such as atrophy of gastric mucosa, hyperplasia of gastric mucosa (localized or polypoid), ulcerative gastritis, syphilitic, tuberculous, or alcoholic gastritis, atony of the stomach or achlorhydria.

CHARLES G. SUTHERLAND, M.D.

Chronic Peptic Ulcers in Children. Howard B. Kellogg. Northwest Med., 38, 129–131, April, 1939.

Kellogg was able to find only 36 reported cases of chronic peptic ulcer in children, adding to this number one of his own. The pathology of these ulcers is similar to that of ulcers found in adults. The location, however, seems to be the reverse. In all these cases, 21 were located in the stomach and 15 in the duodenum.

The symptoms are somewhat less typical in children and may be accompanied by rather alarming conditions, such as severe hemorrhage or perforation. The author states that a large proportion of the patients present a varying degree of pyloric obstruction.

If the possibility of the lesion is suspected, the writer believes that these ulcers will be found more frequently among children.

A. MAYORAL, M.D.

Anterior Pituitary Tumor Associated with Cachexia, Hypoglycemia, and Duodenal Ulcer. Maurice P. Foley, Albert M. Snell, and Winchell M. Craig. Am. Jour. Med. Sci., 198, 1–8, July, 1939.

A case is reported in which a curious aggregation of symptoms, including persistent hypoglycemia, symptoms of cachexia hypophyseopriva, lowered metabolism, an eunuchoid status, and an active and incapacitating duodenal ulcer, was encountered in association with a tumor of the anterior lobe of the pituitary body, which proved, microscopically, to be a chromophobe adenoma.

Several other investigators have reported peptic ulceration associated with intracranial lesions since Rokitansky first suggested the neurogenic origin of gastro-intestinal ulceration in the early 1840's. Briefly, there is clinical and experimental evidence to suggest that acute and chronic peptic ulcers are among the changes produced in the gastro-intestinal tract by intracranial lesions.

The severe hypoglycemia, with the absence of referable symptoms, the lack of response to administration of glucose and epinephrine, and the persistence of the low blood sugar even after removal of a portion of the tumor are noteworthy.

The lowered metabolic rate, common in pituitary cachexia, may represent not only an endocrine effect but secondary changes in nutrition.

BENJAMIN COPLEMAN, M.D.

PNEUMONIA

Lipoid-cell Pneumonia: Adult Type. Henry M. Thomas, Jr., and William F. Rienhoff, Jr. Southern Med. Jour., 32, 1077–1080, November, 1939.

A case of lipoid-cell pneumonia in a 35-year-old white man is reported. This condition is important because it may simulate a tumor of the lung. When suspected, the diagnosis can be confirmed by obtaining the history of the excessive use of oily nasal sprays or drops and by finding lipoid cells in the sputum. In this case, a medicated oily nasal spray had been used for over three years. The difficulty of diagnosis is shown by the fact that this case was mistaken clinically, at operation and at the autopsy table, for a tumor of the lung. Microscopically, there was an interstitial fibrosis with fat droplets in the alveoli, alveolar walls, and lymph nodes.

JOHN M. MILES, M.D.

Lipoid Pneumonia: Report of a Case. Anna M. Young, H. S. Applebaum, and P. B. Wasserman. Jour. Am. Med. Assn., 112, 2406–2409, June 10, 1939.

Lipoid pneumonia (referred to by various authors as oil pneumonia, oil aspiration pneumonia, oil inspiration pneumonia, paraffinoma, paraffin oil tumor, and pneumonolipoidosis) occurs most frequently in infants and in the debilitated old, resulting commonly from the administration of laxative oils or the therapeutic instillation of oils into the nasopharynx with subsequent aspiration of the oily or fatty substance into the lung. Less frequently this lesion has been reported in adults not necessarily debilitated. The authors report a case of the adult type (women, aged 42 years) following repeated aspirations of animal oil, probably cod liver oil, taken by mouth. In addition to the conditions found in the lung similar to those reported by others, metastatic deposits of lipoid material were found, principally in the kidneys and spleen, together with an associated necrotizing arteritis and peri-arteritis.

The lipoid material present in the tissues in this case stained with osmic acid and was saponifiable. Liquid petrolatum stains with scarlet red but not with osmic

acid and is not saponifiable. Animal oil and animal fat (milk, cream, cod liver oil, rabbit fat, and lard oil) produce more severe lesions than do mineral oils. The roentgenogram in this case exhibited the characteristic image of an ordinary pneumonia and not the increased linear markings (fibrosis) or perihilar shadow commonly observed from the aspiration of mineral oil.

CHARLES G. SUTHERLAND, M.D.

RADIATION

Electron Diffraction Studies of Thin Films. I.—Structure of Very Thin Films. L. H. Germer. Phys. Rev., 56, 58–71, July 1, 1939.

Three pages of diffraction rings from evaporated films of some metals and salts are shown in this article. Their interest to the radiologist is the similarity to diffraction rings by x-rays. The wave aspect of the electron has become as commonplace an idea as the particle aspect of light and x-rays. These diffraction patterns by the author, who is a pioneer in electron diffraction, are worth a radiologist's attention, not only for their philosophical import, but for their beauty.

In the experiment related, the author was using them to discover the crystalline structure of very thin evaporated films (2 Å. average thickness to several hundred Å.).

R. R. NEWELL, M.D.

The "Quality" of High Voltage Radiations. Part II.—"Quality" within a Scattering Medium. J. R. Clarkson and W. V. Mayneord. British Jour. Radiol., 12, 168–180, March, 1939.

The authors have measured the effective wave length and the changes in effective wave length which occur in a scattering medium under various conditions. Two methods were used, an emission and an absorption method. The emission method, which is the more accurate, depends upon the difference in photo-electric emission from the walls of metal and carbon ionization chambers with changing wave length; the absorption method depends on the difference in absorption of different wave lengths. The apparatus and procedures are described in detail.

The effective wave length is longer for larger fields and is longer at depths of from 10 to 15 cm. It may be increased to as much as twice the length of the primary. The radiation at the edge of a large field shows half the softening observed in the center. For very small fields this difference is much diminished. These changes are the result of differences in the ratio of scattered to the total radiation. Measurements were made to determine how much of the total radiation was scattered radiation and how much primary at various depths. The absolute amount of scattered radiation reaches the maximum between 2 and 5 cm. below the surface, depending on the wave length of the primary. In general, the scattered radiation makes up about 80 per cent of the total radiation at 14 cm. depth. Variations in the effective wave length of the primary, filtered through water at such a distance that the scattering was negligible, were measured.

The results are presented in the form of graphs. These findings have an important bearing on the interpretation of the selective action of different wave lengths.

SYDNEY J. HAWLEY, M.D.

RADIUM

The Use of Radium in Maintaining a Patent Frontonasal Opening in External Operations of the Fronto-ethmoid Group of Sinuses: A Preliminary Report. Henry L. Williams and Robert E. Fricke. Ann. Otol., Rhinol., and Laryngol., 48, 412–418, June, 1939.

Difficulty in maintaining a patent frontonasal opening is a frequent cause of failure in external operations on the fronto-ethmoid group of sinuses. The authors tried the application of radium post-operatively in 20 cases with apparently good results. The radium was used in the form of a 50-mg. applicator filtered by 1 mm. of brass, 0.5 mm. of silver, and 2 mm. of rubber, for a total dose of 9 gram-minutes. Although the results in this small series of cases were satisfactory, a review of cases not treated by radium seems to indicate that adequate technic in performing the operation is more important than any other factor.

LESTER W. PAUL, M.D.

Individualized Radium Therapy of Carcinoma of the Cervix. H. Reichenmiller. Strahlentherapie, 65, 595, 1939.

The author contends that the success of radiation therapy of carcinoma of the cervix depends largely on the effect of radium. The recurrences can be explained by the rapid drop of radiant energy from the radium applicator toward the periphery. He groups the failures under three headings: cervix cavity carcinoma, in which the tumor grows within the canal leaving the os intact, the huge cervical cancer, and the "spina" recurrence, a carcinomatous mass which develops near the spina ischiadica after the local growth has been successfully destroyed. Reichenmiller outlines his modifications of the standard treatment method which permits higher doses in the periphery, thus affecting cancer cells which otherwise would not receive sufficient dosage. By cauterizing some of the central tumor mass first, the radium screens can be placed closer to the periphery, and, by using longer radium applicators which extend 1 cm. beyond the cervical os. the end-results can be improved definitely.

ERNST A. POHLE, M.D., Ph.D

RADIOLOGY

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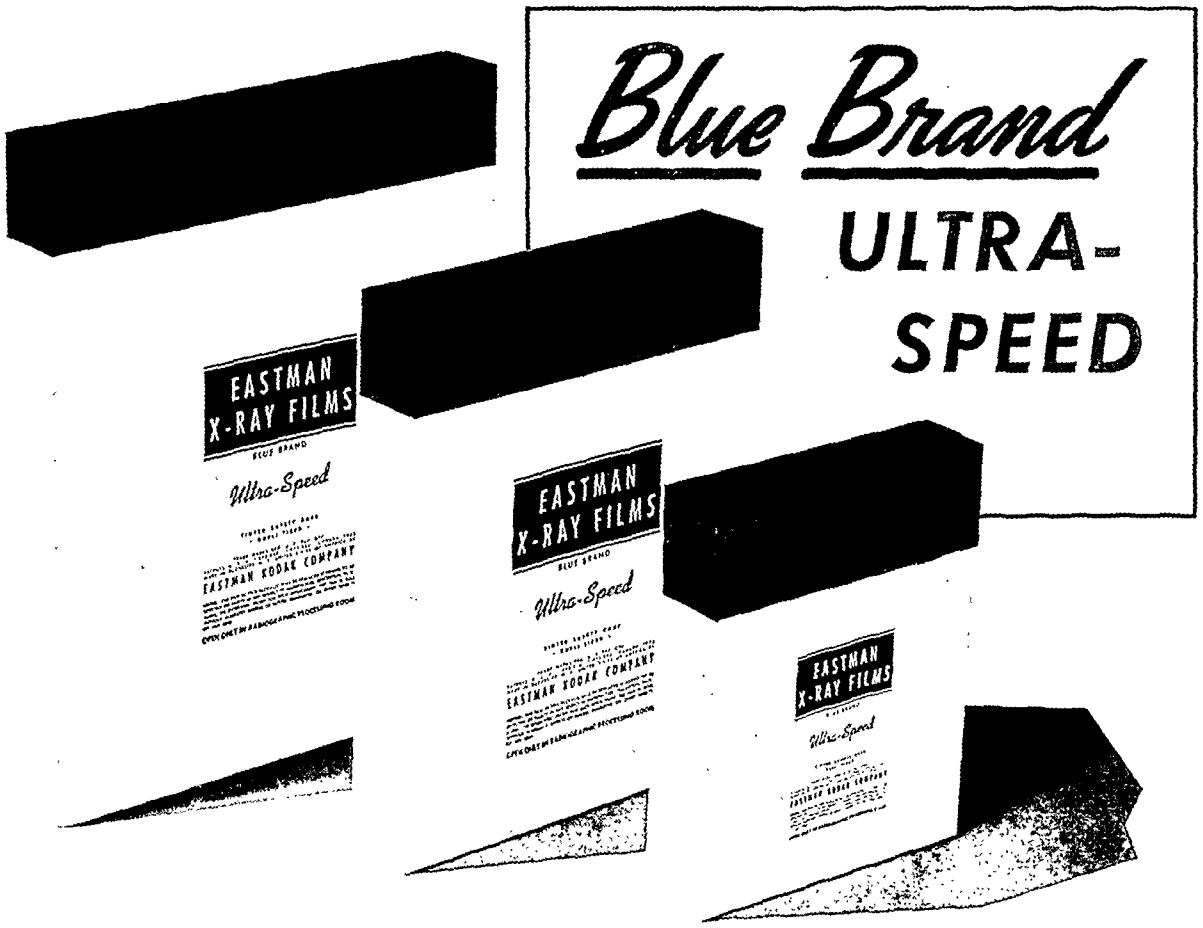
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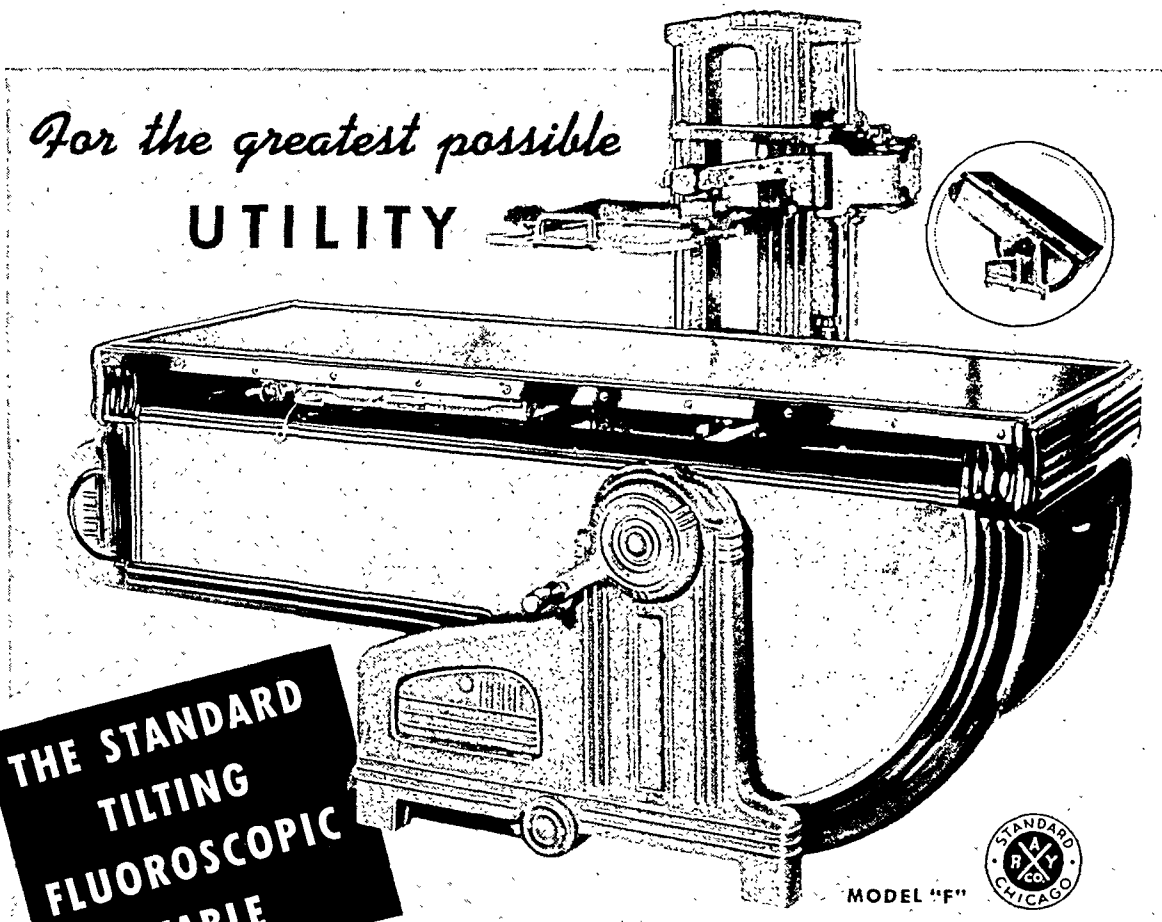
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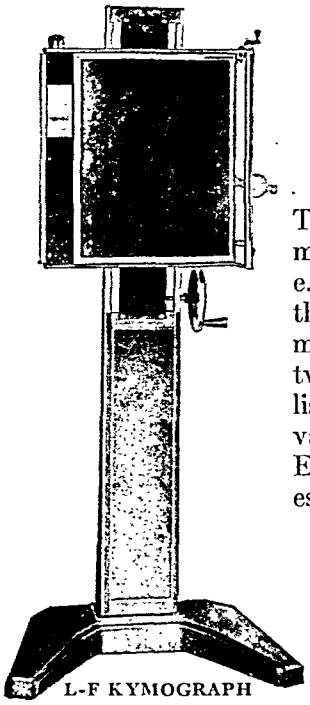
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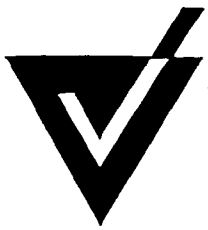
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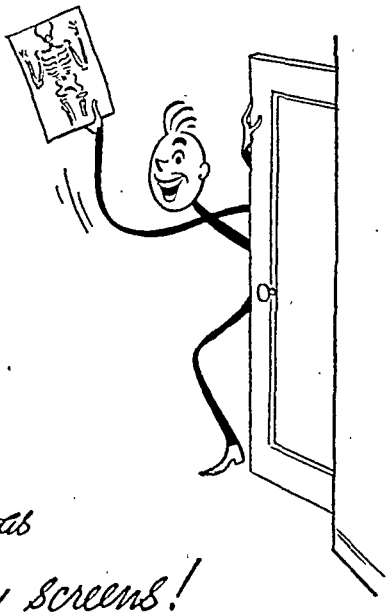
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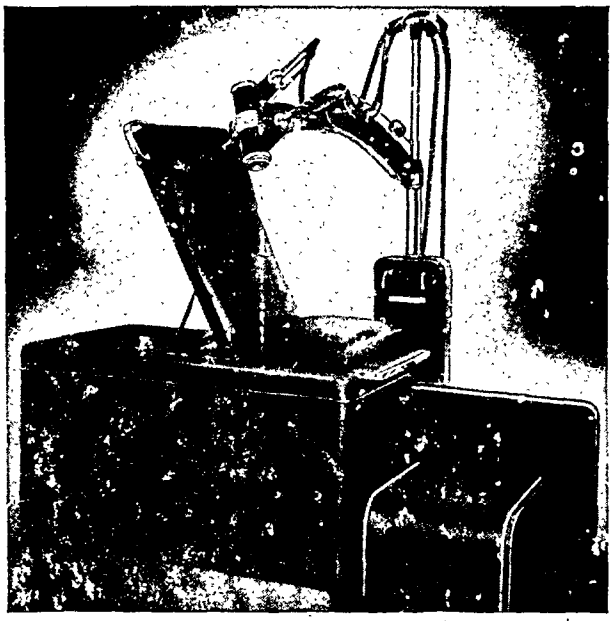
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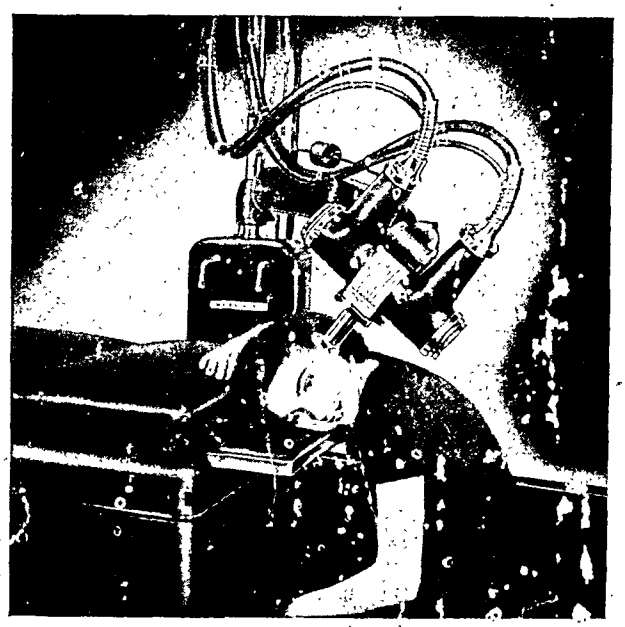
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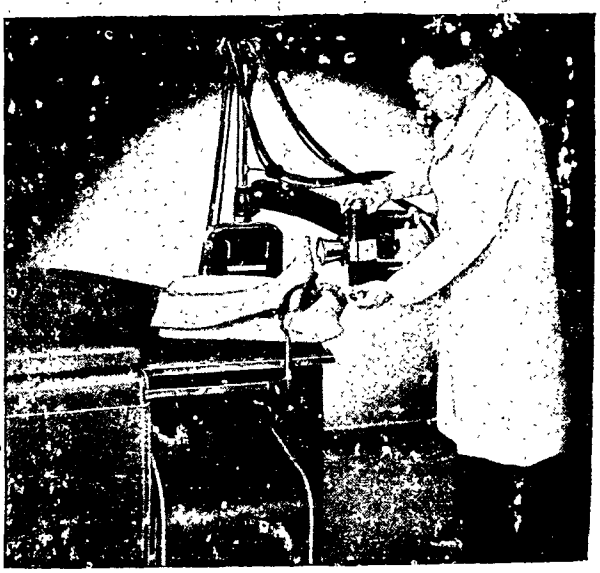


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